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THE ROUND WINDOW—A CRITICAL RE-EVALUATION.*

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Plus ça change, plus c'est la même chose.

History has a curious way of repeating itself in the basic episodes of man's thinking. This is strikingly true in the philosophy of science and medicine.

As scientific knowledge increased especially in the fields of geology, botany, zoology and comparative anatomy, each new step seemed to draw us further and further away from the Old Testament story of Creation. Now a more complete and integrated development of science has proved the truth of the chronology of that story.

The development of atomic chemistry with the atom of each element being the final, immutable and indivisible unit of material and structure seemingly denied the 13th Century philosophic dictum of Thomas Aquinas[1] that all material in the universe has but two attributes—substance and form; that in each material the substance is the same and that any difference lies only in the arrangement of the substance in space; but now as atomic chemistry has evolved into electro-physics we return to sit humbly again at St. Thomas Aquinas' feet.

For some time I have been intrigued, as have all otologists,

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by the efforts made to understand the route and the means by which sound is conducted and processed from the external world to the organ of Corti and thence to the sensorium. The basic fact that we know today concerning sound conduction into the internal ear is that two patent windows must exist. One apparently must give access directly into the cochlea; the other, for practical usable hearing, may open at any other locus into any portion of the perilymphatic space—into the vestibule as with a normal mobile footplate or with a fenestrated, fixed footplate or even into one of the semicircular canals as with fenestra nov-ovalis surgery; but certain facts in all further current theories disturb me.

The round window has been regarded by all modern and semi-modern workers in this field as something of a relief valve for pressures applied via the ossicular chain and the oval window to the perilymph of the scala vestibuli. To accomplish this it is assumed that a given sound wave reaches the footplate in its positive phase and that it reaches the round window in its negative phase or vice versa. Thus the positive pressure at any given point in the scala vestibuli coincides with a negative pressure in the scala tympani at an opposite point along the cochlear partition. As a result the scala media at that spot is displaced downward, and the contained organ of Corti is distorted by torsion and shearing. A rhythmic reversal of these phases then displaces the scala media and the organ of Corti into alternate opposite displacements. The resulting to and fro torsion or bending of the hairs of the hair cells at their distal ends, where they are embedded in the tectorial membrane, produces electrical currents (changes of potential), converting mechanical motion to electrical impulses which finally are interpreted at the auditory cortex as sound.

Many phases of this concept have disturbed me, and I have become intrigued with the idea that the round window plays a greater function in the process of hearing than that assigned by this concept, because no one has as yet, to my satisfaction, substantiated it.

I wondered whether the Creator, by the use of misdirection, much as it is used by sleight of hand artists, had been calling

the attention of investigators to the almost too plausible and too slick ossicular chain system of levers as a conducting mechanism and directing it away from another portal of entry and the true mechanism of the organ of Corti, and whether the investigators had followed this misdirection so persistently that one ingenious explanation after the other had failed.

In a paper read before the Section of Ophthalmology and Otolaryngology of the Southern Medical Association in 1953 at Atlanta[2] I challenged certain phases of it as follows:

"Man being a lunged animal (having evolved from aquatic forms) lives in a medium of air. All sounds brought to him in the ordinary course of his life are brought by waves of alternate condensation and rarefaction in the surrounding air, and his organ of hearing has adapted itself to this change of environmental medium. These waves (see Fig. 1B) are conducted via the external auditory canal to impinge upon the tympanic membrane (see Fig. 1MT) which they set in vibration parallel to their fronts. From the internal surface of the tympanic membrane exactly similar waves are thrown off to traverse the space of the middle ear (see Fig. 1C) to impinge upon the membrane of the round window (see Fig. 1RW). Thus far we have dealt with but one form of conduction, and we will speak of it as aero-conduction.

"Now internal to the round window lies a fluid (man's primordial environmental medium) which in its turn must be set in motion (see Fig. 1). It has greater mass and, therefore, greater inertia than the air of the middle ear, and much of the energy of the air vibrating in the middle ear is lost in setting in motion the membrane of the round window and the fluid beyond it. To offset this loss, a second conducting mechanism has been introduced. This second mechanism is the ossicular chain and it has a dual system of function. It functions first as a system of oscillating levers (see Fig. 1D and D′) cleverly arranged to transform vibrations of relatively large amplitude and low intensity propagated from the vibrating tympanic membrane to vibrations of lesser amplitude but greater intensity and to apply them to the oval window via the footplate of the stapes and its annular ligament. By this means

much of the inertia of the intra-labyrinthine fluid is overcome.

"This manner of conduction by the ossicular chain has one serious obstacle to efficiency—impedance. It is an oscillating system of low mechanical efficiency for conducting sound vibrations. It is suspended by ligaments that have a pillowing action (see Fig. 1P and P'), and it oscillates on ligamentous

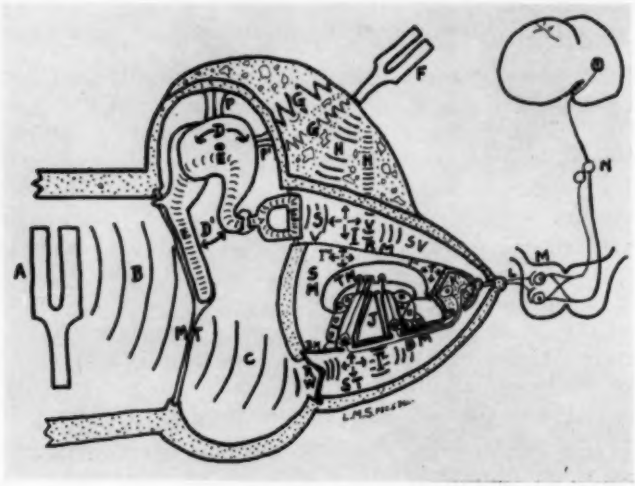

Fig. 1. (A) Source of air conducted sound. (B) Sound waves in the external auditory canal. (B M) Basilar membrane. (C) Sound waves in the air of the middle ear cavity from the tympanic membrane (M T) to the round window (R W). (D and D') Oscillatory motion of the ossicles. (E E E) Molecular vibrations of the ossicles. (F) Source of bone conducted sound. (G and G) Inertial vibration waves conducted by the bone of the skull with intensities varying with the natural impedance of the bone tissue. (H and H) Compression waves via the bone of the skull. (I and I') Hydroconductive waves in the perilymph conducted to it via the round window, the ossicles and the skull bone. Because of the hydrostatic paradox the pressure of these waves is exerted in all directions (arrows). (I''') The still further dispersed waves in the endolymph of the scala media. (J) The convertive mechanism. (K) The spiral ganglion of Corti. (L) The auditory nerve. (M) The dorsal and ventral cochlear nuclei in the pons. (M T) The tympanic membrane. (N) The central auditory tracts. (O) The cortical auditory center. (P) The ligaments attached to the ossicles. (R M) Reissner's membrane. (R W) The round window with its membrane. (S M) The scala media. (S T) The scala tympani. (S V) The scala vestibuli. (T M) The tectorial membrane.

NOTE: This is a Picasso-like effort to present the mechanism of hearing in a single diagram; therefore several aspects of the structures are presented simultaneously. The middle ear is presented in coronal section. The cochlea has been unwound and shortened and the scala media and the organ of Corti have been rotated 90° so as to show them in cross section. The helicotrema is shown although the cross section of the scala media is at its lower end. Reissner's membrane is shown far too close to the tectorial membrane.

bearings that would shock the mechanical sensibilities of a watchmaker who prefers friction-reducing jewel bearings. It is conceivable that such mass oscillations of the ossicular chain may occur at 125 or 250 or even 1,000 cps, but it is inconceivable that the higher frequencies of 4,000 or 8,000 or 16,000 cps can be conducted by such a system.

"If the ossicular chain does conduct these high frequencies

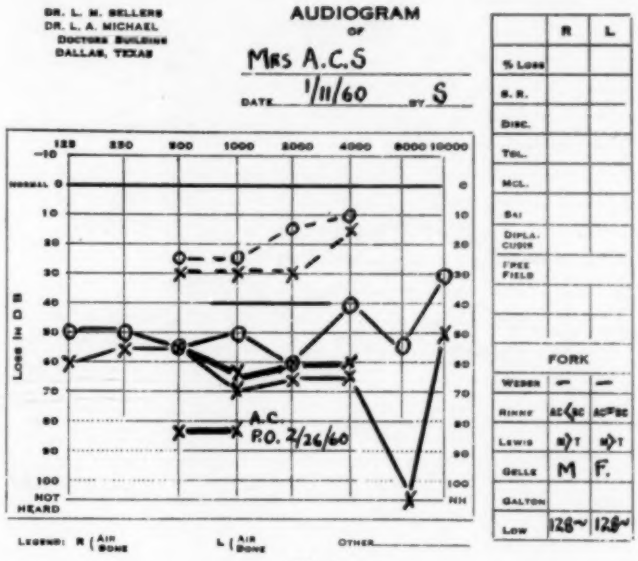

Fig. 2.

* * * then it must be by the second of its dual functions, molecular vibration of the bone tissue itself. Just as the sound of an approaching train is conducted along the rails of the track, from rail to rail across their joints, sound is conducted via the ossicular chain (see Fig. 1EEE). Ossicular conduction then must be both oscillatory and vibratory"; however, even this molecular concept is disturbed by the articular impedances.

In 1955 in my chairman's address to the same Section,[3] I discussed a work on otology written in 1832 by Charles Caswall, a British otologist.[4] Therein I stated, "Basing his conclusions on clear deduction he states unequivocally what I am sure will prove to be the correct answer to a question bemusing otologists today—Does sound enter the labyrinth via the oval window or the round window? His answer is, via both."

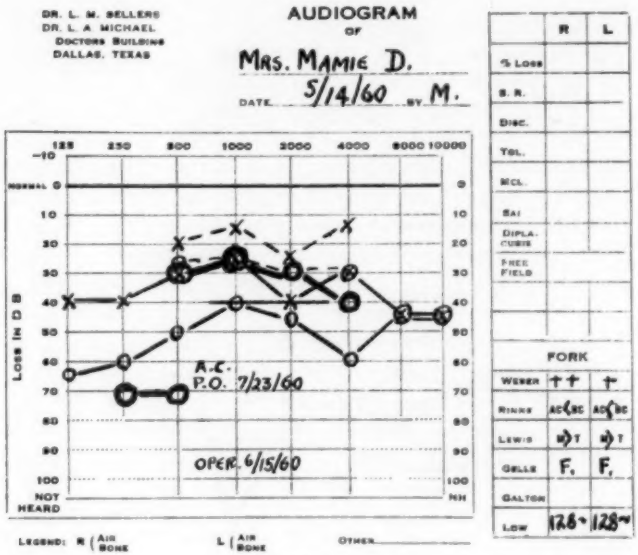

Fig. 3.

Here then is the trail marking the early development of my personal feeling in this matter.

In early 1959 my thoughts began to crystallize when I began the translation of a remarkable book. In the Introduction to this book on the round window written in 1772 by Antonio Scarpa,[5] he stated:

"How often have I considered the anatomical descriptions of the ear [by the great masters], marvelling that all of them with the exception of one or two, have written in detail about

the oval window, the ossicles and the labyrinth but have referred rather carelessly to another true window [the round one] or they have neglected it completely.

"I have assuredly been aware that these structures [just mentioned] have been made matters of great importance, especially by the modern anatomists or the most eminent physicists,

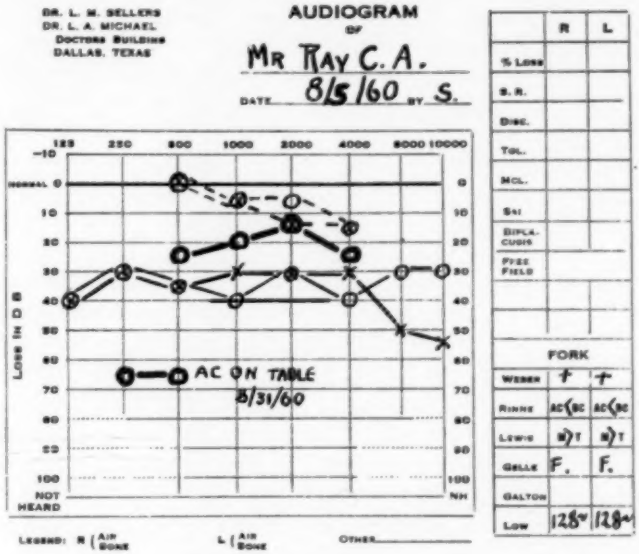

Fig. 4.

in that they maximally conduct and distribute sound into the labyrinth; but the true round window they have depreciated because they say that sounds within the tympanum cannot exit this way, nor can they be so directed, nor is progression of sound in this manner even conceivable.

"So hypotheses concerning hearing are put together which are contrary to the laws of motion and to the actual structures of the ear.

"There is such diversity [of opinion] among these men, as

well as inconsistency and repeated changes of opinion, that one is restrained from acquiescing to opinions of this sort * * *."

He then set about investigating the round window structure showing it to be a canal rather than a simple window, closed at its inner end by the delicate membrane of the round window, which is held in place by an annulus. He showed this membrane to be not a stretched flat one, but one of cone shape, the apex of the cone directed upward and inward, closing off the end of the scala tympani and held taut in place by a bony boss at the beginning of the bony spiral lamina projecting into the opening of the scala tympani. He emphasized the resemblance of these structures to the tympanic membrane at the end of the external auditory canal, their relation to the scala tympani and that sound waves are so directed as to concentrate on the round window membrane. These findings concerning the round window membrane have been confirmed by Bast and Anson[6] (see Fig. 5).

In summation Scarpa stated, "These observations concerning the round window as true in man, quadrupeds and birds, implanted within me the highest suspicion that the function of the round window in hearing was of utmost importance."

He recognized the importance of the ossicular chain and he stated twice that by utilization of the unique structure of the round window "the sound should take two routes to the labyrinth and thus make for better hearing."

I was amazed that my own doubts and my developing view point should have been expressed nearly 200 years previously by the master anatomist Scarpa.

Kobrak in his recent book, although supporting the ossicular chain theory, notes that Bekesy, in measuring electronically amplitudes of vibration of various parts of the tympanic membrane, had found "that for tones above 2400 cps the hammer handle lags behind the adjacent parts of the ear drum membrane."[7] He then notes that the ossicular joints are sliding ones saying, "We have observed in our motion picture studies of fresh human ears that during both the inward and outward motion the incus lags behind the malleus" and "The articulation between the incus and the stapes is a ball and

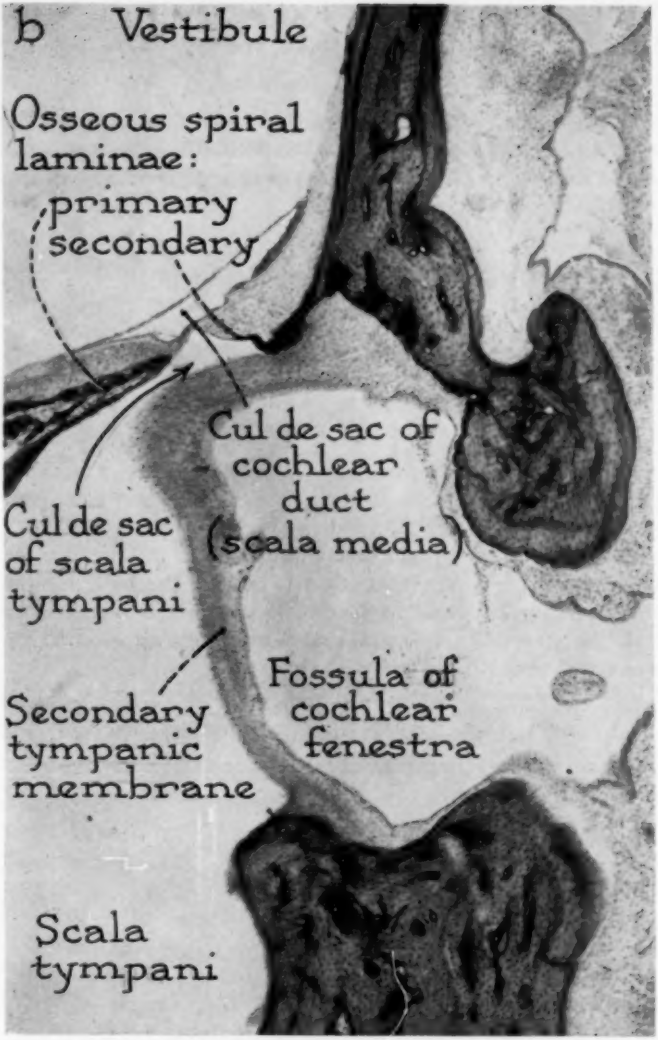

Fig. 5. (The figure is reproduced with the kind permission of Dr. Barry J. Anson from the article by himself and Dr. Bast.[6])

Demonstrating the round window niche, the membrane of the round window (secondary tympanic membrane) lying at the inner end of a canal, and their related structures.

Note how accurately this microscopic section of the human ear demonstrates the gross anatomical description given by Scarpa in 1772. Note well the conical shape of the round window membrane, the apex of the cone directed inward and upward toward the primary osseous spiral lamina roughly duplicating the shape and the arrangement of the tympanic membrane at the inner end of the external auditory canal; note also the **cul-de-sacs** of the scala tympani and the scala media.

The tympanic cavity lies at the extreme right side of the section.

socket arrangement, but the ligaments are weak * * *. The looseness of the incudostapedial articulation prevents the stapes from being pulled out of the oval window in high negative pressures."[8]

He notes the double action of the footplate of the stapes—the foot-tapping action during sounds of lower intensity and the rolling, fixing action (when the stapedius muscle contracts) to prevent injury to the cochlear structures during sounds of great intensity.

Anyone who has attempted transincudal mobilization can testify to the weakness of this joint.

Here we have a system said to conduct high frequency sound that may be understood (although only approximately so) by anyone who has ridden the rear car of a steam drawn train with the old type loose couplings. His memory of the jolt experienced as the train began motion in one direction only, taking up the slack at each of the couplings, will remain vivid. Compound this with having the train attempt to reverse its direction repeatedly and rapidly.

May we mention a few clinical observations that cause us to look with something less than enthusiasm on the ossicular chain as the prime organ of conduction of *all* sound frequencies from the tympanic membrane to the scala media.

In a successfully performed fenestration with *complete disruption* of the chain, the residual loss of hearing has been calculated mathematically as 30 db[9] and found clinically as 20 db. This loss which is of such small volume, unless appearing precipitately, is rarely noted in any individual since following surgery it permits "normal usable hearing." Lempert has reported at least one case in which the hearing loss level was 10 db for the speech frequencies following fenestration,[10] with removal of the incus and head of the malleus.

At the October, 1960 meeting of the American Academy of Ophthalmology and Otolaryngology, Rambo reported (but not yet published) a new procedure for fenestration of the horizontal semicircular canal for the relief of otosclerosis without an open cavity. In this procedure he does not cut a skin flap

for closure of the new window. A flap of temporal muscle hinged anteriorly is turned backward to fill the attic, antrum and mastoid. The incus is removed, the malleus is left intact, and the muscle flap is laid over the new fenestra, interposed between it and the head of the intact malleus. He assumes (mistakenly, I feel) that this mass of muscle with its inordinately high impedance transmits the vibrations (or more actually the oscillations) of the head of the malleus to the perilymph of the scala vestibuli (and so to the organ of Corti) via the new fenestra which it contacts. Here clearly the portal of entry must be the round window with the new fenestra acting as a relief valve under the pad of transplanted muscle.

These same objections apply to the procedure of Hans Heerman of Essen, reported at the Detroit International Symposium on Otosclerosis on November 12, 1960, in which after removal of the stapes and enlargement of the oval window, impedance-laden layers of fascia and fat are interposed between the open oval window and the end of the long process of the incus as the link between the disrupted ossicular chain and the scala vestibuli. Certainly this impedance-laden mass of tissue cannot transmit the high frequencies perceived in the successfully operated cases.

Recently Rosen has become awakened to the lessened importance of the chain as the prime sound conductor. He has reported a respectable series from his own cases and those reported by others[11] in which hearing reached the zero or 10 db level after finding disruption of the chain at operation or after simple fenestration of the footplate with the entire stapes remaining fixed rigidly in the oval window. These findings are equally surprising with the loss of the functioning chain if it is the prime conductor of sound.

Kobrak, honest scientist that he was, concludes that "The construction characteristics of the sound conducting system of the middle ear results in its being less than high fidelity."[12]

Some amazing ideas have been presented regarding the functions of the round window aside from that of a relief valve. Experimental work done on blocking the round window artificially has been inconclusive and contradictory; but the

idea has been advanced that this structure plays little or no part in hearing, an idea that the most ardent oval windowites do not support; however during transmeatal surgery I am sure that we all have observed typical otosclerotic hearing type conductive changes that could be ascribed only to interference with the round window. Three such cases from our own practice are of interest here.

In the first case the audiologic findings were those of true otosclerosis of the oval window area (see Fig. 2); however, after mobilizing the footplate without surgical accident, no motion at the round window niche with correct methods of observation could be observed and no improvement in hearing resulted.

In the second case again findings of typical otosclerosis were found audiologically (see Fig. 3). On June 15, 1960, transmeatal mobilization on the right ear was attempted. The middle ear was found to be criss-crossed with firm adhesions which enwrapped the ossicles and involved both windows. The stapes was freely movable. The adhesions were cleared away as completely as possible except from the niche of the round window without change in hearing. These latter were then approached with temerity because of at least one bad operative result which we felt had been due to surgical trauma of the round window membrane. As the niche was cleared of adhesions the effect on the patient was electrifying because of sudden hearing improvement. An audiogram at this time confirmed this immediate improvement. The improvement has been maintained.

The third case gave equivocal evidence of clinical oval window otosclerosis (see Fig. 4). Here exploratory tympanotomy was advised and was performed on August 31, 1960. The footplate was found freely mobile, but the round window niche was occluded by a dense opaque fibrous membrane stretched across its opening. As soon as this was removed the tinnitus stopped, and the hearing improved as shown by the audiogram taken during surgery. This improvement has been maintained.

In 1958[13] we published a series of cases of apparent per-

ceptive type deafness relieved by catheterization that could be explained clinically only on the basis of temporary round window occlusion. The number of cases in this series has increased since that publication.

Here we offer possible explanations of these cases. The surgical cases presented partial occlusion of the round window by membranes semipermeable to sound. The cases relieved by catheterization were more completely blocked to sound transmission by more completely occluding plastic plugs of thick mucus.

As we pass beyond the oval window our difficulties with the ossicular chain—oval window—scala vestibuli theory increase.

The compression waves rushing up the scala vestibuli must funnel through the narrow helicotrema and come rushing down the scala tympani always in opposite phase at each point along the organ of Corti to impinge against the round window membrane which must move outward with each inward movement of the footplate.

This probable misconception is amplified by the clinical observation of reciprocal motion of the stapedial footplate and the round window membrane produced by surgical manipulation of the stapes.

I have chosen Hallowell Davis' Biophysics and Physiology of the Inner Ear[14] as an authoritative digest of recent work in this field for source material; however, he begins his discussion of "Problems of Auditory Physiology" with the statement, "We will *take it for granted* that the movements of the stapes give rise to movements of the sensory structures of the organ of Corti and that the movements somehow set up the nerve impulses."[15] This assumption we feel provides at best a questionable foundation for an interpretation of the experimental work on the cochlea which follows, but first we feel that we must mention two preliminary facts in reference to most of the experimental work which has been done.

The closed cavity of the tympanum and adnexa is a necessary resonating chamber. It is almost a *sine qua non* of nor-

mal hearing. Of course we have seen patients with tympanic membrane perforation who have quite adequate hearing providing all other structures are uninjured; but if in an otosclerotic patient with successful fenestration of the canal and restoration of normal usable hearing the tympanic membrane is ruptured post-operatively destroying the integrity of the tympanum as a *closed* resonating chamber, the effect on that restored hearing is disastrous. We must now remember that all observations made directly on ossicular chain action and on round window movements in response thereto are made on a badly damaged tympanum—completely unroofed, with a large myringopuncture or with the entire labyrinth removed so as to view the inner faces of both the oval and round windows. The resulting changes in the functions of the conducting mechanism as a source of error must be considered.

The same criticisms may be levelled at visual observations made to date on intracochlear wave motions. The cochlea must be opened changing a contained fluid system (with due regard for the aqueductus cochleae and aqueductus vestibuli) to an open one with consequent profound changes in action.*

With these two reservations let us return to consider some of the experimental work reported.

In discussing the decreasing stiffness (there is no tension) of the basilar membrane as measured by Bekesy[16] as the distance a locus ascends the cochlea, the following illuminating statement is made although its true significance seems to have been overlooked, "Furthermore it is not Reissner's membrane or the tectorial membrane, but the basilar membrane that determines the movements of the cochlear partition."

Your essayist must call to your attention that the basilar membrane is in direct contact with the scala tympani—round window system rather than the ossicular chain—oval window —scala vestibuli—Reissner's membrane system.

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*A method of entering both the scala vestibuli and the scala tympani in the living animal without loss of perilymph or disturbance of the tympanic membrane, the ossicular chain or the middle ear as a resonating chamber has been devised by Dr. Daniel Martinez of the Texas University Southwestern Medical School and the Veterans Administration Hospital both of Dallas. Investigation of phase relationship electronically is being developed by these routes.

In another experiment Bekesy[17] found that no areas of motion of the basilar membrane for frequencies higher than 1,600 cps could be observed with the microscope. This would seem to limit the frequency of vibration of the footplate arising from ossicular chain oscillations capable of sound transmission. This raises the probable limit of 1,000 cps that I had set roughly for such action in 1953, but it remains far below the 4,000, 8,000, 10,000 and 16,000 cps to which the cochlea responds clinically.

At this point does the ossicular system pass from the process of sound transmission as the dominant system and does the perceptive organ begin to bring into play some system that can respond to pressure waves in the fluids of the internal ear that are so minute that the response is in the order of electronic motion rather than molecular vibration?

Bekesy also noted travelling waves in models with the stapes and a round window each variously located[18] (and note that these are waves of compression and not undulation) which always travelled from the narrow, stiff end of the dividing membrane to the broader end, no matter what the location of the driver (stapes). This resulted in these travelling waves travelling *toward* the stapes if it were located at the broader end of the dividing membrane. He confirmed these findings in the cochlea by introducing the sound via an opening drilled at the apex.

Wever and Lawrence[19] confirmed this by applying acoustic energy at 5,000 cps at the stapes and at the apex. The conclusion is "like Bekesy, from these and other observations that stimulation of the cochlea by any channel, *i.e.*, by oval window, by a hole in the apex and by bone conduction, always produces the same pattern of vibration in the cochlea"; but apparently it did not occur to them to attempt stimulation via the round window.

Does this not suggest some function for the footplate other than, or in addition to, the function of the input of sound waves?

Davis agrees that up to a certain point transmission of the

energy of sound stimulation is via the fluids. After that it is via the basilar membrane.[20]

Davis concludes his discussion of these experiments by Bekesy and by Wever and Lawrence with this[21] illuminating statement ". . . the present reviewer, when lecturing about the pattern and the dynamics of cochlear movements, refrains from shaking a rope to illustrate a travelling wave and he avoids motion pictures that make an analogy with waves of the sea running up a sloping beach."

The essayist here interposes that there are two factors militating against the theory of alternating up and down motions of the organ of Corti resulting in a shearing action of the tectorial membrane across the hair cells with consequent bending of the hairs of these cells.

One factor is that the physical consistency of the entire organ offers too great an obstacle to its acting thus, *i.e.*, to rise above the resting position to a peak and then to return beyond the resting position to a negative peak with a return again to the resting point (a simple sinusoidal wave) with the rapidity it requires to produce a pure tone in the higher frequencies. The organ *in toto* is rather jelly-like in its consistency, and to the writer it is inconceivable that the segment corresponding to the stimulating tone should be so acting, while neighboring areas remain relatively quiescent or are responding to different frequencies.

The second factor is that any pressure wave so acting from the scala vestibuli would exert its force not directly on the organ of Corti but on Reissner's membrane. If such a force acted on Reissner's membrane it would not be communicated to that portion of the organ of Corti lying below it, but would be disseminated instantly throughout the scala media according to the law of the hydrostatic paradox, *i.e.*, that any pressure applied at any point on a contained fluid instantly exerts the same pressure throughout the fluid and consequently on all walls of the container (see Fig. 1I‴′).

Now granting that the ossicular chain—oval window—scala vestibuli route is the route of sound entry, the above objec-

tions might be explained away by using a pure tone, *i.e.*, a simple sinusoidal wave as is used apparently in all the experiments under consideration; but we do not live in a world of pure tones. The note "a" (440 cps), sounded on the oboe as a standard by which the members of a symphony orchestra tune their respective instruments, is recognized by our ears as emanating from the oboe and is distinguished from the same note then given forth by all the other instruments. This individual "timbre" is produced by the harmonics or overtones peculiar to each instrument, so that now the single note of 440 cps produces a very complicated wave. Just how do these waves emanating from the various instruments sounding simultaneously arrange their courses up the scala vestibuli and down the scala tympani so as to be always in difference phase? Is not this concept baffling? Now let the orchestra *tutti* sound a major or a minor chord, each choir of instruments sounding harmonizing notes of different frequencies. This becomes much more baffling. Now let there come forth a blue chord in which there is faulty mathematical relationship between the note frequencies. Just how can the pitiable fluid system of the cochlea acting on the organ of Corti from above and in exact difference phase from below escape the worst of frustrations when handed such a preposterous and impossible task?

"Come now, and let us reason together, . . ."[22]

Could not these apparently clashing data, as revealed by experimental scientists, be reconciled if the sound pressure waves could be applied directly to the basilar membrane at specific spots under the hair cells and against a tectorial membrane made for the moment more resistant to motion? This resistance can be conceived as a result of counter pressure exerted upon it by increase of pressure in the scala media communicated to it via Reissner's membrane from pressures communicated to the scala vestibuli by movements of the stapedial footplate.

In 1952 Bast and Anson described a *cul-de-sac* coming as a finger-like projection from the scala tympani which interposes itself between the end of the scala tympani *per se* and the

membrane of the round window as if to assure contact of this scala with the round window membrane so that vibrations of the latter would communicate themselves to the perilymph of the scala tympani. Another *cul-de-sac* comes similarly from the scala media to end blindly in the vestibule. These seem to offer double assurance of these scalae's being in contact with the pressure waves originating in the areas of their blind endings[6] (see Fig. 5).

Such a concept does not denigrate the functions of the ossicular chain. The craftsman's anvil against which his hammer strikes is fully as important as is his hammer. How much more so this would be if his anvil could be made to vary its resistance so that soft gold and tough iron could be manipulated simultaneously with greater facility, accuracy and safety. In addition to its acknowledged function as a conductor of low frequencies the footplate of the stapes with the action of the intratympanal muscles does just this, saving the delicate structures of the organ of Corti and possibly those of the cristae and maculae from damage by excessive positive or negative pressure. It may also modify the quality of the tone as the left hand of a string instrument player by its rocking motion produces a vibrato. Finally it definitely provides the final refinement of hearing which closes the gap between 20 db loss and normal hearing.

This concept most assuredly has been implemented by a communication by Dohlman to the Southern Section of the American Laryngological, Rhinological and Otological Society at its 1958 meeting.[23]

By auto-radiography Dohlman demonstrated within the fluids of the tectorial membrane and the cupulae, the presence of a protein-bound potassium-muco-polysaccharide complex. He noted that on motion of these fluids there was more rapid motion of the positive K ion producing an electrolyte of positive potential. Within the hairs of the hair cells which fitted into canaliculi of the tectorial membrane and cupulae he noted the tissue juices of the hair cells. These carried a negative electrical potential. The surface membrane of the hair acted as a dielectric causing the cell itself to act as a condenser. As

the hairs were projected more tightly into and against the walls of the canaliculi the dielectric effect was lessened, and a negative electric current potential was set up. As they were withdrawn a positive electric current potential was set up. As they alternated between insertion and withdrawal an alternating current potential developed just as in a cochlear microphonic.

When I heard how a simple up and down motion of the basilar membrane of exceedingly fine amplitudes could produce cochlear microphonics I stated, in an unreported discussion of this paper when it was read, that no longer would otology, like general world history, be unable to pin point the exact moment that the dark ages ended and the Renaissance began.

For Otology (if this work is substantiated), this I felt was it.

It must be realized that the highly complex wave patterns impressed by even this simplified mechanism are beyond the capability of the organ of Corti to integrate into musical chords or to analyse into component notes. This function must be reserved for the central nervous system in some such pattern as the substantia reticularis determines the direction, rate, amplitude and duration of nystagmus once it is evoked by the hair cells of the cristae.[24,24A,24B]

This is a plea by a practicing otologist rather than an experimental scientist for a re-evaluation of the round window. The writer feels that it is in this region that much future progress in intratympanic surgery will occur. Much constructive work remains to be done on this subject. As yet we have no clear cut idea of what symptoms and signs a disability in this area may produce. In some of the cases of disability in the round window area that have been cited here a conductive deafness manifested itself. In others a true perceptive syndrome was evoked.

House in 1959[25] reported hopeful results in surgery of the round window, but one year later, after further experience with the procedure, he revised his conclusions[26] and abandoned this work until better surgical techniques could be devised.

In the meantime Ibanez,[27] in a procedure which he calls sonoinversion, is utilizing the round window as a portal of entry in tympanoplasty when the ossicular chain is destroyed and the oval window function is disturbed. Here by roughening the edges of the round window niche he attaches remnants of the tympanic membrane to these edges at the same time attempting to create a pocket in communication with the ostium of the Eustachian tube. In cases of otosclerosis, by combining this procedure with either fenestration of the horizontal canal, or with removal of the footplate of the stapes and covering the open window with a vein or skin graft, he feels that his results are improved.

Now a final word. Can it be that we are completing another cycle that resembles those that have gone before? Is all the fine and accurate work done by great investigators, devoting their lives to answering the question of how we hear, much of which seems to be contradictory and which refuses to mesh into a coherent whole, about to fall into place? Are we about to return to Scarpa as we have returned to the first two chapters of Genesis and to St. Thomas Aquinas? Are we again about to implement Alphonse Karr's *bon mot* concerning French politics as it may apply to Simple Scientific Truth:

"Plus ça change, plus c'est la même chose—
The more it changes, the more it is the same thing."

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EXPERIENCES WITH CONSERVATION SURGERY IN LARYNGEAL AND PHARYNGEAL CARCINOMA.*†

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INTRODUCTION.

If sacrifice of the larynx is the only surgical treatment for large laryngeal and pharyngeal cancer, this leaves no consideration for sparing the function of the larynx in these patients. The term, conservation surgery, is reserved for partial laryngectomy and pharyngectomy operations in the treatment of the more malignant forms of supraglottic and pharyngeal cancers where total laryngectomy is the most widely used form of therapy today. For glottic cancer the principles and techniques of the laryngofissure, hemilaryngectomy procedures, including variations of the anterior commissure methods, are well established and represent a form of conservation surgery.

Conservation procedures require accurate clinical and laryngographic localization as well as knowledge concerning what structures must be left for a functioning larynx and pharynx. The senior author has previously presented several methods for operating upon tumors on the epiglottis and in the pyriform sinus.[1-4] The problems concerning diagnosis have been presented in a previous article.[5] The incidences of cervical lymph node metastases from epidermoid carcinoma of the larynx and their relationship to certain characteristics of the primary tumor have also been published.[6]

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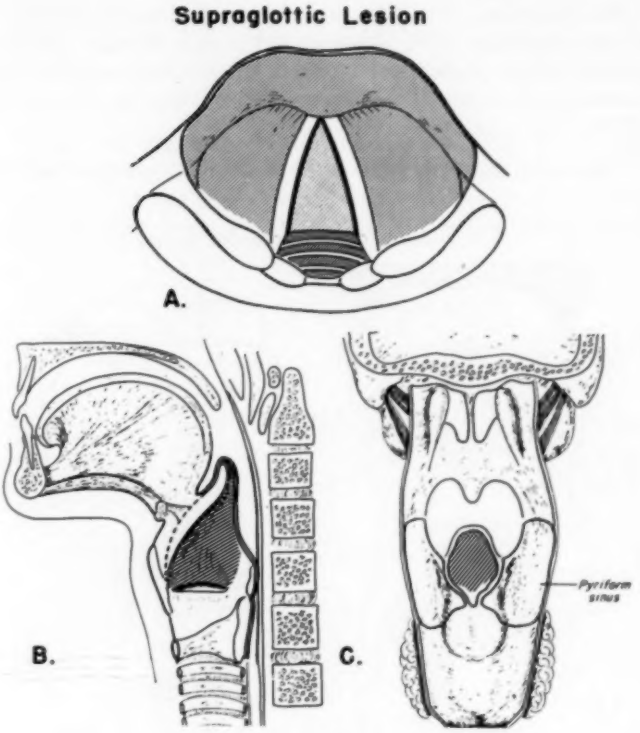

Fig. 1.

In this paper, the postoperative complications and detailed pathologic study of the primary lesion with its relationship to lymph node metastases and follow-up will be given.

ANATOMIC CLASSIFICATION.

This classification scheme is not intended to supplant or replace any existing nomenclature. With the hope of better understanding behavior of lymph node metastases, an arbitrary division between the supraglottic and pharyngeal tumors was made in an endeavor to relate nodal metastases with location of the primary carcinoma.

"Supraglottic" (see Fig. 1) refers to the area from the tip of the epiglottis to its fixed portions and includes the free borders of the false cords. Although epiglottic tumors may extend to the lateral free border of the epiglottis, the aryepiglottic folds should be free.

"Superior hypopharyngeal" (see Fig. 2) anatomically in-

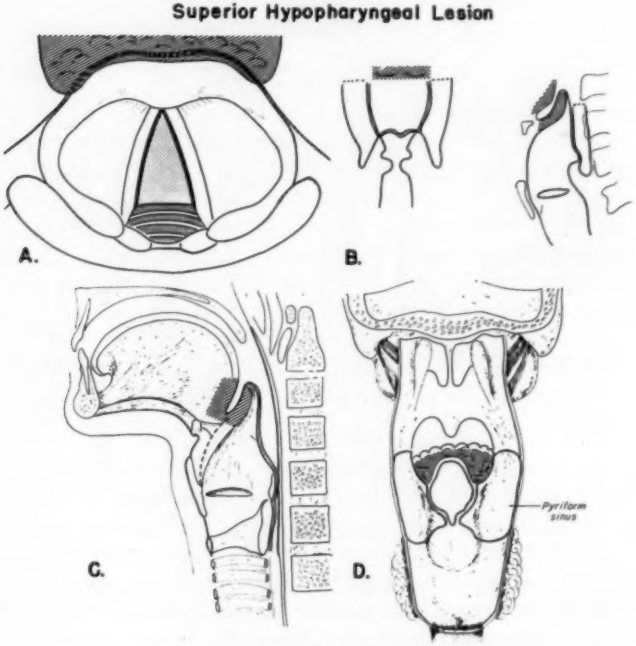

Fig. 2.

cludes the lingual surface of the epiglottis, the vallecula and the "base of tongue." It includes the glossoepiglottic folds and is confined to an area inferior to the V-ring of the circumvallate papillae of the tongue, which theoretically is the true superior margin of the base of the tongue. Obviously a tumor that extends from the circumvallate papillae to the

vallecula would represent a huge tumor and would not be suitable for a conservation operation. The senior author has included these tumors originating in this area under the term "extended supraglottic subtotal laryngectomy procedure."[7]

"Posterior pharyngeal wall" tumors (see Fig. 3) are those in the zone between the base of tongue (base of tonsil) and the arytenoid and medial to each lateral wall of the pyriform sinus. Tumors in this area are infrequent and often involve

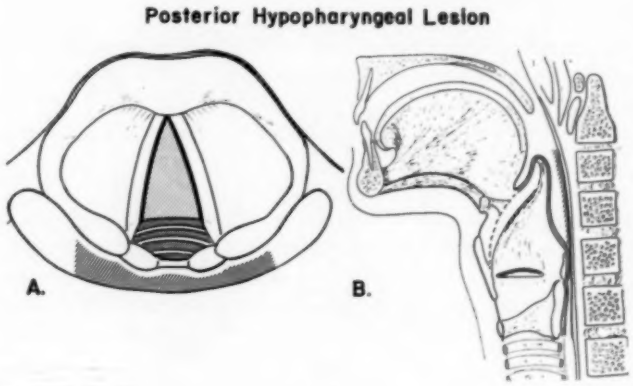

Fig. 3.

the glossoepiglottic fold or, minimally, the area of the superior hypopharynx or pyriform sinus. Since their metastatic behavior is not unlike either group, they are included in the superior hypopharynx.

"Pyriform sinus" cancers (see Fig. 4) are really hypopharyngeal tumors. These may be defined as lesions in the area of the lateral hypopharynx bounded by the glossoepiglottic fold superiorly, the arytenoid and apex of the pyriform sinus inferiorly, laterally the thyroid cartilage and medially by the aryepiglottic fold and anterior pyriform sinus wall. The medial border is lateral to the free border of the epiglottis. Posteriorly these are limited by an ill-defined vertical line at level of the arytenoid where the lateral wall becomes the

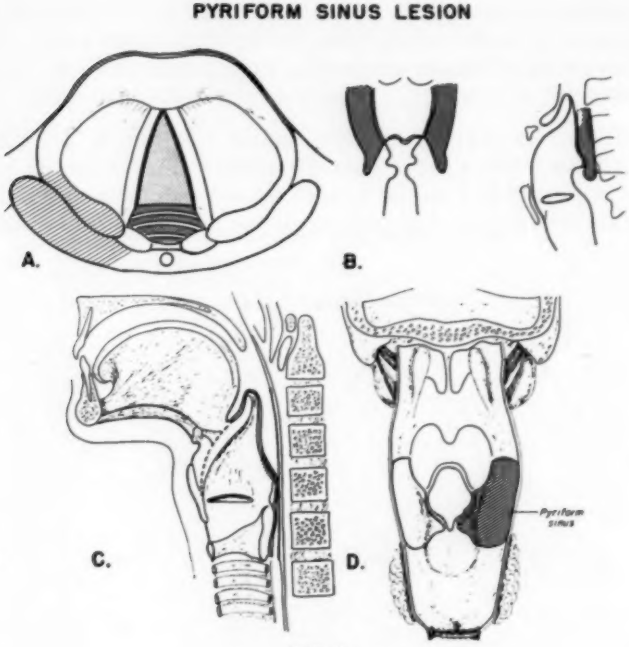

Fig. 4.

posterior wall of the hypopharynx. Since the aryepiglottic fold is the medial wall of the pyriform sinus, tumors of this structure are grouped with pyriform sinus neoplasms.

DIAGNOSIS.

It seems needless to point out that successful extirpation of tumors in these areas depends on accurate clinical assessment of the extent of the primary lesion. Therapy is not undertaken without biopsy proof of carcinoma. Biopsies of lymph nodes suspected of containing carcinoma were not done.

Supraglottic.

Such localization is possible by indirect examination or direct laryngoscopy, but most important of all, by the use of the laryngogram. If the tumor is within the area from tip to

the petiole of the epiglottis and a free zone of uninvolved tissue is visible by laryngogram, a wide surgical margin may be obtained in such a case. When tumor extends to the false cord, and only a narrower zone free of tumor can be expected, consent for possible total laryngectomy must be obtained prior to operation. The vocal cord must be freely movable, and the anterior commissure must be uninvolved by carcinoma.

Superior Hypopharyngeal.

Tumors in this area will be restricted to the lingual surface of the epiglottis, the vallecula and the inferior base of the tongue. They are usually more to one side or other and occasionally associated with other separate lesions of the hypopharynx. Palpation of the base of the tongue will aid in determining the extent of invasion of the tongue by carcinoma.

Posterior Pharynx.

These tumors are confined to a zone between the base of the tongue and the level of the arytenoid. Direct examination is necessary to determine the lower level, and freedom of the laryngeal structures should be present. Tumor fixation to the vertebral bodies must be absent.

Pyriform Sinus.

Lesions of the pyriform sinus or fossa are in general easily visualized by indirect and direct examination and correlate well with pathological and laryngographic findings. When the tumor is infiltrating the larynx and has fixed the arytenoid, the only treatment is total laryngectomy or more extended procedures that require resection of the pharynx and esophagus. One notable "blind area" for visualization is the anterior portion of the pyriform sinus. It is considerably more difficult to determine within the confines of the pyriform fossa, how much of the pharynx and parts of the larynx may be resected with adequate margin and preservation of useful function.

The tumor must spare the post cricoid area and preferably not involve the apex of the pyriform sinus. For medial wall lesions, the tumor may extend to but not impair arytenoid

motion. Superiorly, it should not extend much above the glossoepiglottic fold to be suitable for partial operation. It is important to stress that prior to operation, the thyroid cartilages should be demonstrated to be intact.

SURGICAL APPROACH.

The essential principles for exposure of the pharynx and larynx are outlined in previous articles by the senior author. Consent for total laryngectomy should be obtained if there is reasonable doubt about the probability of being able to carry out conservation surgery. In some instances, exploratory phar-

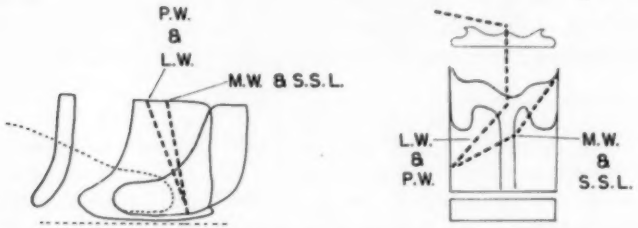

Fig. 5. Area of cartilage and bone transection. The amount of transverse thyroid cartilage section required for lesions of the supraglottic and superior hypopharyngeal lesion (SSL) and medial wall of the pyriform sinus (MW) is the same. One third the distance from the thyroid notch to its inferior border.
Less extensive cartilage section is required for lesions of the lateral wall of the pyriform sinus (LW) and the posterior wall (PW).

yngotomy at the conclusion of the neck dissection is necessary to determine whether the operation can be performed.

General Principles.

No detailed account will be given for the radical neck dissection since numerous papers have been written on this subject. Incontinuity surgery is maintained with the primary area. The neck dissection is completed before surgical entrance is made into the hypopharynx. The muscles over the larynx are transected transversely over the thyroid cartilage down to the perichondrium at each superior border of the cartilage. The perichondrium is reflected inferiorly to allow exposure of the thyroid cartilage.

The point of transection of the cartilaginous structures of the larynx will vary according to location of the primary lesion. The cartilage is cut with a Stryker saw prior to entrance into the pharynx. For supraglottic lesions and superior hypopharyngeal lesions the thyroid cartilage is cut transversely at a level between its upper anterior third and lower two thirds (see Fig. 5). For pyriform and posterior pharyngeal wall lesions, an oblique section of one thyroid cartilage allows direct exposure (see Fig. 5). The hyoid bone is divided

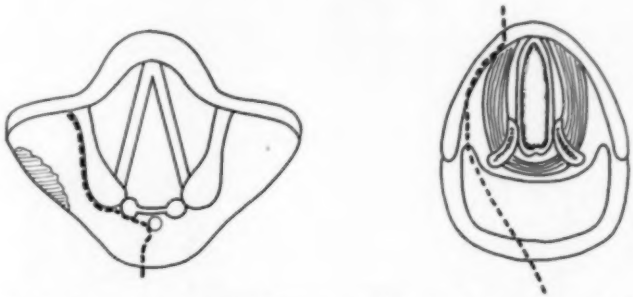

Fig. 6. Lateral wall of pyriform sinus lesion. The area of resection of the thyroid cartilage and hyoid allows soft tissue excision along the aryepiglottic fold and part of the posterior wall of the hypopharynx.

in the midline or more toward the normal side and the hyoid bone removed with the lateral pharynx. Removal of part or all of the epiglottis can be performed in lesions of the superior hypopharynx, lateral wall (pyriform sinus) or medial wall or for posterior wall as each case dictates (see Figs. 6, 7, 8).

In resection of the base of tongue, one lingual artery may be sacrificed, but not both. Thick split thickness skin grafts are no longer used for supraglottic lesions or superior hypopharyngeal lesions. Skin grafts are necessary for posterior wall lesions and for large pyriform sinus lesions.

The following areas of anatomical resection are compatible with preservation of function of larynx and pharynx (see Fig. 9):

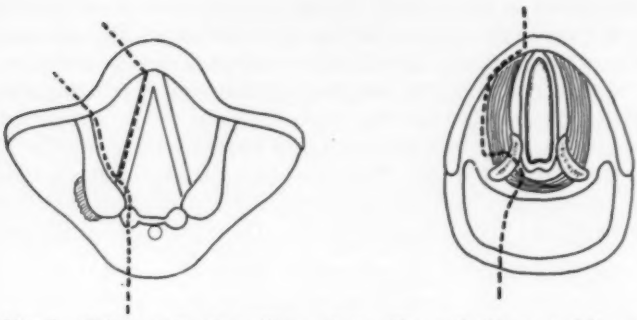

Fig. 7. Medial wall of pyriform sinus. The soft tissue excision will include the epiglottis, false cord, arytenoid (except for the vocal process) and lateral wall of the pyriform sinus.

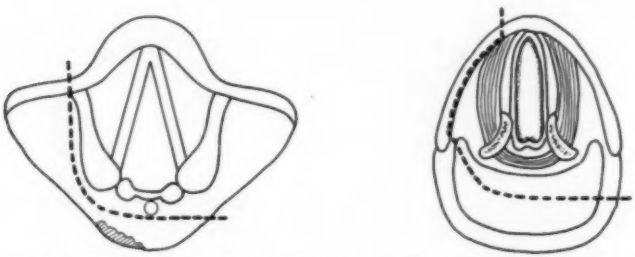

Fig. 8. Posterior wall lesion. The soft tissue excision includes the lateral wall of the pyriform sinus, but none of the components of the larynx. The thyroid cartilage and hyoid bone are removed with the specimen.

1. Removal of the entire epiglottis, false cords, ventricle and aryepiglottic folds; *2.* removal of one arytenoid and pyriform sinus; *3.* removal of the posterior pharyngeal wall and part of the cricopharyngeus; and *4.* removal of the posterior third of tongue.

Complications of conservation surgery.

A total of 58 patients was operated upon, with an immediate operative mortality of three cases (five per cent). It must be stressed that in the selection of these cases, poor risk patients are included. Many of these patients had previous coronary occlusion, lowered pulmonary reserve or advanced age, or other geriatric degenerative diseases. Coronary throm-

Anatomic Zone of Resection with Conservation Surgery Compatible with Preservation of Function

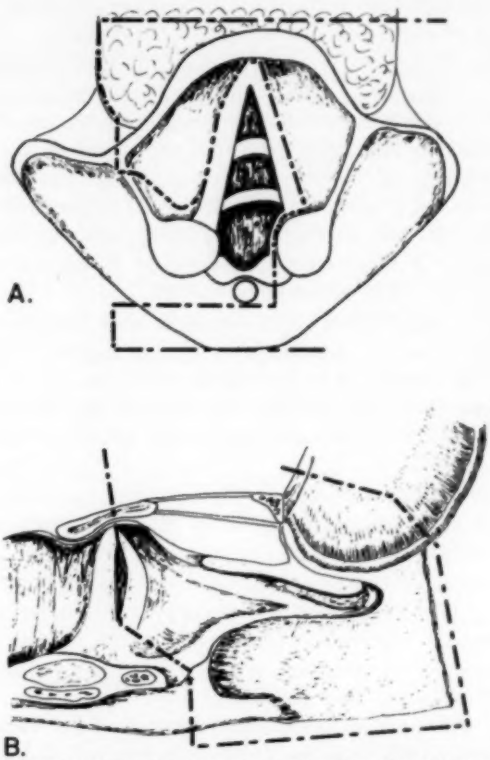

Fig. 9. The dotted line represents the borders for the widest margin of resection for tumors within this area that is consistent with preservation of deglutition and voice.

bosis, cerebral hemorrhage and gastric ulcer bleeding accounted for the three deaths.

Ordinary oozing of blood usually ceases in one-to-two days. If bleeding is brisk, a pharyngeal pack may be kept in place for one-to-two days. The use of stents minimizes the bleeding.

Most of the immediate pulmonary postoperative complica-

tions are mild and are of the nature of increased secretions from the tracheotomy or occasionally bronchopneumonia. Antibiotic therapy is not a routine in our postoperative orders. Of the 58 patients, pulmonary infarction occurred once, and this patient survived.

Originally in the reconstruction of the hypopharynx a musculo-fascial flap (strap muscles) with a skin graft was used. This was occasionally complicated by fistula formation. The technique has been modified by suturing the external perichondrium of the thyroid cartilage to the base of the tongue. At the present time no skin graft is used for epiglottic resections. Fistula formation has been markedly reduced. In one instance a staged pedicle flap was necessary to close a large postoperative pharyngeal defect.

Fistula formation is more common with excision of pyriform sinus lesions compared with supraglottic operations. Five of 22 supraglottic operations and five of 13 pyriform sinus cases developed fistulas. Except for one instance, which required pedical flaps, the remaining fistulas have spontaneously closed.

Serious wound infection with resultant perichondritis in the remaining larynx occurred in one instance. Secondary total laryngectomy was necessary.

The degree of function retained by conservation surgery is directly related to extent of surgery. All epiglottic cancers and superior hypopharyngeal lesions and lateral pyriform sinus lesions with resection of the tongue and epiglottis have excellent function. Two structures are related to good deglutition, the arytenoid and cricopharyngeus muscle. Total arytenoid removal, including the false cord but not the true cord, seems to impair the degree of swallowing function. Semisolids and liquids are easily managed. One occasional persistent complaint is the difficulty with mucus which requires frequent clearing of the throat. Skin grafts are important for large excisions since they minimize distortion and twisting of the larynx during healing.

When the arytenoid and the posterior pharyngeal wall have been removed down to the cricopharyngeal area, swallowing function may be somewhat altered. Of the present ten cases,

TABLE I.

CONSERVATION OPERATION
SUPRAGLOTTIC CARCINOMA
(SUPRAGLOTTIC SUBTOTAL LARYNGECTOMY)
(Not including base of tongue, vallecula, pyriform sinus, pharynx, hypopharynx, etc.)

Entire series: 22 cases. 20 male, 2 female. 22 white.
21 Epidermoid ca., 1 adenocarcinoma.

Epidermoid ca: 21 cases. 19 male, 2 female. 21 white.
16 epiglottic, 1 false cord, 4 both.

Average: 57.1 years (36-74)
Average size: 2.6 cm. (0.5-4.0)*

	Nodes Negative	Nodes Positive	No Neck Dissection	Total
Number	15	4	2	21
Average size (cm.)	3.0 (0.5-4.0)	1.7* (1.0-2.5)		2.6* (0.5-4.0)
Differentiation:				
Well	1	0		2
Moderate	8	1		9
Poor	6	3		10
Invasion:				
Nerve sheath	0	0		0
Angio‡	0	1		1
Cartilage (epigl.)	10†	2		14†
Soft tissue (pre-epiglottic)	9	2		12
Ca-*in-situ*:				
About tumor	4	0		4
At excis. margin	0	1		1
Invasive ca. at excis. margin	1	3		4

16 elective neck dissections, tumor present in 2 (12.5%)
5 therapeutic neck dissections, tumor present in 4 (80%) (2 bilateral neck dissections)

21 total neck dissections, tumor present in 6 (28.6%)

*Size of two (2) tumors unknown.

†In only two (2) cases was there cartilage invasion without soft tissue invasion.

‡Further studies with elastic tissue stains in progress.

three had some difficulty handling solid foods. Two were subsequently improved by section of the cricopharyngeal muscle in a secondary operation. It is now our practice to cut this muscle at the initial operation. In one patient, only semisolids and liquids can be managed.

Cine-fluoroscopic studies in cases with impaired deglutition show a pseudo-diverticulum created at the junction of the skin

TABLE II.

CONSERVATION OPERATION
SUPERIOR HYPOPHARYNX
(EXTENDED SUPRAGLOTTIC SUBTOTAL LARYNGECTOMY)
POSTERIOR PHARYNX
(PARTIAL PHARYNGECTOMY)

(Vallecula, base of tongue, hypopharynx, lingual surface of epiglottis, etc. Not pyriform sinus or pure epiglottis.)

13 cases: 12 male, 1 female; 13 white.
13 epidermoid carcinoma.

Average age: 58.4 years (46-77)
Average size: 2.7 cm. (0.5-4.0)*

	Nodes Positive	Nodes Negative	Total
Number	7	6	13
Average size (cm.)	2.3 (0.5-4.0)	3.3* (3.0-4.0)	2.7* (0.5-4.0)
Differentiation:			
Well	0	2	2
Moderate	5	3	8
Poor	2	1	3
Invasion:			
Nerve sheath	1	1	2
Angio†	1	1	2
Cartilage (epigl.)	3	2	5
Soft Tiss.—Pre-epig.	1	1	2
Soft Tiss.—Neck	1	0	1
Ca-*in-situ*:			
About tumor	2	2	4
At excis. margin	3	1	4
Invasive Ca. at excis. margin	0	0	0

8 elective neck dissections, tumor present in 4 (50%)
9 therapeutic neck dissections, tumor present in 5 (55%) (2 bilateral neck dissections)

17 total neck dissections, tumor present in 9 (52.9%)

*Size of three (3) tumors not known.
†Further studies with elastic tissue stains in progress.

graft and upper esophagus by upward movement of the larynx in the first act of deglutition.

The character of the voice is remarkably good with supraglottic and superior hypopharyngeal lesions, except perhaps when the arytenoid cartilage is exposed. This will lead to lateral fixation of the cord and hoarseness will result. The voice is quite hoarse when the entire arytenoid is removed, but excellent for posterior wall and lateral wall lesions.

Temporary tracheotomy is necessary for every conservation operation. Permanent extubation was successfully accomplished in every supraglottic, superior hypopharyngeal and posterior wall and lateral pyriform sinus lesion within a one month period. In some instances, successful decannulation has been possible as early as two weeks after the operation.

TABLE III.

CONSERVATION OPERATION
(PARTIAL LARYNGOPHARYNGECTOMY)
(PRYIFORM SINUS including lateral and medial wall of pyriform sinus)
13 cases. 3F, 10M. 10 right side. 3 left side. Avg. age 59 (43-81).
Avg. size of lesion 2.9 cm. (2-4). All had neck dissections unilateral.

Number of patients:	+ Nodes	- Nodes	Total
13	12	1	13
Differentiation:			
Well	0	0	0
Moderate	6	0	6
Poor	6	1	7
Invasion:			
Nerve	5	0	5
Angio*	2	0	2
Soft Tissue	1	0	1
Thyroid Cart.	2	0	2
Thyroid	0	0	0
Carcinoma-*in-situ*:			
About tumor	4	0	4
At Edge	0	0	0

One had invasive carcinoma at margins.

4—3 with + nodes had 2nd or simultaneous carcinomas.

One patient (+ nodes) had persistent carcinoma with subsequent laryngectomy.

10 therapeutic neck dissections, tumor present in 10 (100%)
3 elective neck dissections, tumor present in 2 (66%)

*Further studies with elastic tissue stains in progress.

In five pyriform sinus cases where arytenoid removal was done, an average of a month was required for permanent extubation of the tracheotomy tube. Usually the delay was because of fistula formation or difficulties in re-establishing deglutition. In one of five, a two and one half months' delay was necessary because fistula and skin loss required reconstruction procedures to the pharynx and larynx.

Late local recurrence will be discussed later.

Results.

Of the 58 conservation operations performed, the study reported here concerns only 48 cases performed at the Washington University Group of Hospitals. A detailed review (see Tables I, II, III) and re-examination of the specimens and followup was available. Ten other partial operations were performed at other hospitals within the city and are excluded from this study pending re-examination of the specimens.

TABLE IV.

INCIDENCE OF NODAL METASTASES

	Number of Cases	Cases with Nodal Metastases
Supraglottic	21	4 (19.0%)
Superior Hypopharynx and Posterior Hypopharynx	13	7 (53.8%)
Pyriform Sinus	13	12 (92.3%)
Total*	47	23 (48.9%)

$X^2_2 = 17.3$ $P < 0.0005$

*One case of adenocarcinoma not included.

Division of these lesions into the three groups can be justified by one of the pathological findings: the incidence of nodal metastases as shown in Table IV. The difference in incidence of nodal metastases in the three groups is statistically significant ($X^2_2 = 17.3$: $P < 0.0005$). There is no real difference in the other pathologic findings in the three groups (differentiation of the tumors, invasion of nerves and vessels, etc.).

DISCUSSION.

Selection of cases on the basis of the primary lesion located within an anatomic zone considered amenable for conservation surgery yielded a random group. These lesions were classed according to their location. No effort was made to select only fungating tumors for this operation. Tumor size was a factor only if it extended outside of the field considered amenable to conservation surgery. Since neck dissection was routinely

performed, except in the first two cases, the lesion could be correlated with nodal metastases.

Supraglottic lesions were infrequently found to have unsuspected metastases, 12.5 per cent compared to 28.5 per cent for the overall metastatic rate. Originally, lesions of the lingual surface of the epiglottis were included with the supraglottic group; but we found that these tumors behave like other pharyngeal lesions with respect to their metastatic behavior, and they belong with the superior hypopharynx class. The neck dissection does not increase morbidity or mortality and facilitates closure of the pharynx in that the loose pharyngeal tissues could be easily mobilized. It is possible that, with accumulated experience, in view of the low yield of unsuspected metastases, elective neck dissection for pure supraglottic lesions will not be necessary.

Probably because of the small number of elective neck dissections in the superior hypopharyngeal and pyriform sinus groups, the difference in percentage of unsuspected metastasis in the supraglottic group compared with the other two groups is not statistically significant. ($X^2 = 2.24$ with Yates' correction, $P = 0.14$).

It is significant that 11 of 22 supraglottic cases have passed the two year plus survival level (see Table V). It is known that any case surviving past the second year without recurrence has a very high probability rate of surviving five years without disease.[8] This compares favorably with the 40 per cent survival rate in our slightly more extensive epiglottic carcinoma where total laryngectomy has been performed.[7,8]

The total lack of local recurrences in the remaining larynx is significant when one notes that in one half of the 22 cases the lesion was in the inferior portion of the epiglottis where a narrow zone of surgical margin was inevitable.

The superior hypopharyngeal and posterior wall lesions, as a group, are intermediate in their metastatic rate between the epiglottic and the pyriform sinus cancer. Inapparent metastatic rate was 50 per cent compared to an overall metastatic rate of 52.5 per cent. Late local recurrences at the surgical

TABLE V.

SHORT TERM RESULTS

Supraglottic	5 yr.	3	
	4-5	2	
	3-4	3	11/22 (50%) over 2 yr.
	2-3	2	11/22 On inf. epiglottis or
	1-2	3	inf. vest. lesion.
	<1	4	
		17	
Dead		5	Adenocarcinoma
			Pulmonary metastases (+ node)
			Coronary thrombosis—late
			Gastric hemorrhage
			Second lung cancer
Superior Hypopharyngeal and Posterior Wall Lesion (3)			
	2-3 yr.	3	
	1-2	1	3/13 (23%) over 2 yr.
	<1	3	
		7	
		1	alive with persistent disease
Dead		5	1 local recurrence tongue
			1 recurrence opposite node
			1 coronary thrombosis postop.
			1 cerebral hemorrhage postop.
			1 pneumonia late
Pyriform Sinus	4-5 yr.	2	
	3-4	1	
	2-3	0	3/13 (23%) over 2 yr.
	1-2	1	
	<1	5	
		9	
		1	alive with disease
Dead	3	1	secondary carcinoma
		1	local recurrence superior margin
		1	local recurrence inferior margin (cartilage destruction)

margin resulted in one instance. There is an overall survival for two plus years in three of 13 (23 per cent).

Pyriform sinus lesions proved to have the highest metastatic rate. In 12 of 13 cases, lymph nodes contained metastatic tumor, with an inapparent rate of 67 per cent compared to 92.3 per cent overall metastatic rate. Three of 13 cases survived two plus years. If one combines all of the pharyngeal

lesions together, one has 6/26 cases (or 23 per cent) surviving thus far without tumor. This compares favorably with the author's 25 per cent survival for total laryngectomy.[7,8]

Infrequent local recurrences with partial laryngectomies are significant. There were only two local recurrences in the pharyngeal cancer group; these were at the surgical margin, and in neither was tumor seen at the margin on microscopic examination of the specimen. Carcinoma *in situ* was found in 13 of 47 cases which gives an index as to the carcinogenic potential of the remaining pharynx. In one of the local recurrences, the thyroid cartilage destruction was overlooked at the initial operation and accounted for postoperative persistence of tumor in spite of irradiation. (Secondary total laryngectomy was done in one instance of pyriform sinus cancer.)

In the author's experience the incidence of local persistent tumor in the conservation group is no higher when compared to local persistence of tumor with hypopharyngeal carcinoma where total laryngectomy and pharyngectomy are performed.

The arbitrary separation of the primary lesion according to anatomic location has yielded important differences from the surgical, functional, and pathologic aspects. Surgical approaches for these lesions vary considerably, and the physiologic results attained are dependent upon the degree of resection. Arytenoid removal results in a poorer voice, and surgical margins that involve the cricopharyngeus impair the efficiency as to the kind of food that can be swallowed.

Fistulas are more common with pyriform sinus lesions and less common with supraglottic lesions. All conservation operations have more problems than the more easily executed operation of total laryngectomy.

Lack of local recurrence for supraglottic cancer and its infrequent metastases, and the uncommon local persistences of cancer with the superior hypopharyngeal and pyriform sinus cancer correlate well with the ultimate short term survival. The survival is good for supraglottic cancer and promising for the pharyngeal cancers, comparing well with those

lesions in the same location treated by total laryngectomy in the author's experience.

CONCLUSION.

Separation of lesions according to location yielded distinct differences between tumors restricted to the supraglottis and those of the rest of the hypopharynx.

Short term results of the epiglottic lesions appear very encouraging.

It is significant that the high survival rate for the supraglottic is related to the low metastatic rate when compared to the remainder of the pharynx.

Short term results for other hypopharyngeal lesions appear encouraging. For a lesion known to carry a poorer prognosis, the survival of this group of unselected cases appears encouraging.

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EARLY DIAGNOSIS OF HYDROPS OF THE LABYRINTH.*

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The symptom complex manifested by vertigo, tinnitus and deafness was originally described in detail and with considerable accuracy by Ménière:

> "Attacks of sudden vertigo, nausea and vomiting are quite common. I have seen a large number of them. There is, however, a perfectly distinct group of such patients, the specific nature of which is characterized by loss of auditory sensitivity. . . . If those who might have the occasion to observe the sudden onset of vertigo, nausea and vomiting in their patients would take the pains to examine carefully the auditory apparatus, if they measured the degree of hearing in both ears, if they would question their patients as to what had taken place in these organs since the appearance of these 'congestive accidents of the brain,' they would recognize that the inner ear is the site of the lesion, the point of origin of the symptoms which had previously appeared so dangerous to life, and against which they had employed a rigor of treatment as uncomfortable to the patient as it was futile, a violent therapy of which not the least important inconvenience is not only to harm the patient but also to cause him to doubt the intelligence of his physician."

Ménière described the deafness present as a nerve type, with greater inability to hear bass voices than treble. He stated that the disturbance might involve both ears, but that more commonly, only one was involved.[1]

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For many years the disorder was called Ménière's symptom complex or Ménière's disease.

It was not until 1938 that Hallpike and Cairns[2] described the pathologic findings as an edema or hydrops of the cochlear duct. These findings have been verified by Lindsay[3] and others. Over the period of years the disorder has become to be known as *hydrops of the labyrinth.* This is indeed fortunate since the term refers to a specific physio-pathologic entity.

The purpose of this paper is threefold: *1.* to demonstrate that the hearing defect due to hydrops of the labyrinth can be accurately differentiated from other types of deafness; *2.* to demonstrate that defective hearing frequently is the first symptom of hydrops of the labyrinth; *3.* to emphasize the importance of early diagnosis of hydrops of the labyrinth in Aviation Medicine.

There are certain definite characteristics of the symptoms and findings which make the diagnosis fairly reliable in well advanced cases.

VERTIGO.

The vertigo which accompanies hydrops is typically of the whirling type. Objects in the field of vision appear to be moving to the right or left. The vertigo is usually so severe that the patient seeks the recumbent position but even then is not comfortable. It is aggravated by movement. In many instances the patient is incapacitated and actually falls. Some individuals have had auto accidents when the episode came on while they were driving, and became incapacitated before they could stop the car. The episode lasts for minutes or hours but not for days or weeks. The vertigo is accompanied by nausea and when severe, by vomiting. Nystagmus usually accompanies the vertigo. The episode may terminate as suddenly as it began, or it may gradually disappear.

The episodes have a tendency to recur at irregular intervals. In the early stages the episodes may be months or years apart. Because of the long remissions McNally and Stuart[4]

decided that five years would be the minimum period free of vertigo before a patient could be classed as "cured," regardless of whether or not the patient was receiving treatment. As time goes on the interval between episodes tends to become shorter.

TINNITUS.

The tinnitus is fluctuating in character and is frequently worse preceding or during an attack of vertigo.[5] The tinnitus may remain between attacks. It is typically described as a "roaring" or buzzing sensation which simulates the sound of the sea or a seashell. It is a white noise rather than a pure tone.

DEAFNESS.

The deafness tends to increase and decrease in the early stage. One ear only is affected at first; later both ears may be involved. Between attacks the hearing may return to normal. The hearing may fluctuate independently of the vertiginous episodes. Wright[6] pointed out that the deafness may precede any other sign of the disorder by months and years. There is a tendency for greater interference with the transmission of low tones than high; this being more apparent on the bone conduction curve than on the air conduction curve.

Tonndorf[7] has conducted experiments on a cochlear model in an effort to find an explanation for the type of hearing loss incurred in the early stages of endolymphatic hydrops. The model was so constructed that the endolymphatic fluid volume could be increased. When the fluid volume was increased, Reissner's membrane showed its maximal distention in the center section because of its uniform stiffness and fixation at both ends. The basilar membrane formed its maximal distention near its distal end where stiffness was least. The distention per unit distance was reciprocally related to stiffness which varied in an exponential manner. The frequency response curve of the model was then determined. When the endolymphatic pressure was increased in discrete steps, the loss in sensitivity increased with each incremental step of the

endolymphatic fluid volume. He concluded, "Such models, upon increase in endolymphatic pressure, displayed: *1.* a loss in sensitivity; *2.* a shift of the location of maximal displacement amplitude along the cochlear partition toward the base of the cochlea (both of the phenomena were more pronounced for low than for high frequencies); *3.* harmonic distortion. These observations are paralleled by clinical findings which are generally accepted as being characteristic of endolymphatic hydrops; hearing loss predominantly for low tones; diplacusis; and lowering of the threshold for aural harmonics. Recruitment and the disproportionally large speech perception loss which also are observed clinically cannot be explained on the basis of the present experimental data."

Diplacusis is likewise characteristic of hydrops. The pitch of a pure tone sounds higher in the diseased ear. This may be due to the change in maximum amplitude displacement of the basilar membrane as shown in Tonndorf's cochlear model. The change always occurred in the direction of the base of the cochlea—in the direction of higher frequency localization.

Wright[6] termed the type of deafness found in hydrops as "cochlear deafness" to distinguish it from the loss of hearing seen in degeneration of the fibers of the auditory nerve or spiral ganglion. Recruitment, which may be described as an abnormal growth or increase in loudness, is characteristic of cochlear deafness. Dix, *et al.*,[8] related recruitment to hair cell pathology in 1948. It is present in patients with hydrops during the period of time when the deafness is present; it is also present in cochlear deafness due to other causes. Steinberg and Gardner[9] pointed out in 1937 that if the threshold of the normal ear be raised or shifted by means of a masking noise, a recruitment-like increase in loudness occurs as the intensities of pure tones are increased above their shifted thresholds. Reger[10] has confirmed this observation. Recruitment is best measured by Fowler's test. It must always be measured by alternate, never by simultaneous stimulation of the two ears.[11] Cochlear deafness may also be determined by difference limen tests. Jerger, *et al.*,[12] have recently described a unique method for the clinical assessment of differential intensity discrimination which they refer to as the "short

increment sensitivity index (SISI)." They found that conductive and retrocochlear losses showed very low SISI scores, never exceeding 15 per cent. Ménière's (hydrops) and noise-induced losses yielded relatively high percentages, the lowest score obtained being 70 per cent.

Low discrimination scores are characteristic of hydrops of the labyrinth. Walsh[13] has stated that the discrimination score is seldom more than 50 per cent. The score is much lower than would be suspected from the slope of the threshold audiogram or the hearing level. In spite of the fact that the relationship between the low discrimination scores and hydrops of the labyrinth was reported in 1943,[14] this test is not so frequently used as it could be, possibly due to lack of amplified speech equipment in most otologists' offices.

Low discrimination scores are also obtained in the presbycusis of senior citizens. The age and lack of fluctuation in the hearing and absence of the characteristic tinnitus differentiate presbycusis from hydrops. Patients who have a severe hearing loss for pure tones in the speech range with a sharp drop off in the audiometric curve for the speech frequencies may also have low discrimination scores. In these patients this finding is readily explained by the slope of the pure tone audiogram. Patients with an acoustic neuroma likewise have low discrimination scores.

The continuous and interrupted Békésy audiograms have been useful in the differentiation in site of lesion in perceptive deafness. When hydrops is present the continuous audiogram shows a smaller amplitude in the tracing than does the interrupted although both curves nearly parallel the pure tone threshold. With an acoustic neuroma the interrupted Békésy audiogram shows a normal pattern consistent with the pure tone audiogram. The continuous Békésy audiogram shows the inability of the ear to maintain response under sustained stimulation. As a result there is a progressive elevation of threshold over a period of time.[15]

LABYRINTHINE TESTS.

Tests of labyrinthine function should be performed even

though no characteristic findings may be expected. The response to caloric testing may reveal the affected labyrinth to be normal, below normal or nonfunctioning.[16,17]

CASE PRESENTATIONS.

The following abstracts of case histories and examinations demonstrate in one way or another typical features of hydrops of the labyrinth. In most instances the patients were examined one or more times elsewhere before they were referred to the School of Aviation Medicine, USAF. For the sake of brevity, only air conduction audiograms are reported. In all patients the bone conduction paralleled the air conduction thresholds.

Case 1. W.T.B., Captain, Navigator, Age 29. In 1954 the patient had a constant roar in the right ear of one month's duration. In March, 1958, the roaring recurred, and he had lightheadedness for 30 minutes. In June, 1958, the tinnitus was more marked, he had true vertigo and noted deafness for the first time. The patient was driving and had to stop because of the vertigo which lasted about 30 minutes. There was complete recovery, including the deafness, in two weeks. The patient noted a progressive hearing loss between August, 1958, and June, 1959, although he had no other symptoms.

Hearing Tests: A review of audiograms between September, 1957, and June, 1959, revealed fluctuating hearing, perceptive type, in the right ear.

AUDIOGRAM—June 18, 1959.

	250	500	1000	2000	3000	4000	6000	8000
Right Ear:	35	60	50	15	5	30	30	20
Left Ear:	–10	–10	–5	–10	–5	5	25	–5

Speech reception threshold—Right Ear: 34 db.

Discrimination score—Right Ear: 52 per cent at 80 db. The normal range of discrimination scores in our laboratory is 75 to 85 per cent.

Diplacusis and recruitment present. Maximum comfort level: 60 db on right.

Caloric test—Right: Increased sensitivity. Left: Normal sensitivity.

Case 2. E.J.B., Captain, Senior Navigator, Age 41. The patient had roaring tinnitus and deafness intermittently from 1952 to 1954. Between March and May, 1954, these symptoms were followed by vertigo, nausea and vomiting. One episode occurred during flight, and a student navigator had to take over. In February, 1957, he had another similar episode.

Hearing Tests: Audiograms accompanying the patient revealed defective hearing in March and April, 1957, right ear, more marked for the low frequencies than the high frequencies.

AUDIOGRAM—May 8, 1957.

	250	500	1000	2000	3000	4000	6000	8000
Right Ear:	10	10	5	5	10	20	35	40
Left Ear:	–5	–5	–5	–5	–5	5	–5	0

Speech reception threshold—Right Ear: 3 db. Left Ear: –7 db.

Discrimination score—Right Ear: 90 per cent. Left Ear: 92 per cent.

At present all tests are within normal limits.

Caloric test—Right: Normal. Left: Normal.

Case 3. R.E.B., 2/Lt., Pilot, Age 23. In June, 1956, the patient had intermittent episodes of defective hearing, right ear, followed by a disturbance of equilibrium of five to six hours' duration. In August, 1956, he developed tinnitus. In December, 1956, and January, 1957, he had whirling vertigo, decreased hearing, nausea and vomiting. Between February and September, 1957, he had one episode of deafness, right ear, without other symptoms.

Hearing Tests:

AUDIOGRAMS—September 17, 1956.

	250	500	1000	2000	3000	4000	6000	8000
Right Ear:	45	40	35	25			20	30
Left Ear:	–5	–10	–5	0			–10	–10
				February 4, 1957.				
Right Ear:	0	0	5	–5	–5	0		–10
Left Ear:		No change						

Speech reception threshold—Right Ear: –2 db. Left Ear: –3 db.

Discrimination score—Right Ear: 86 per cent. Left Ear: 90 per cent.

Tests at this time within normal limits.

Caloric test—Right: Normal. Left: Normal.

Case 4. A.B.C., 1/Lt., Pilot, Age 24. The patient developed roaring tinnitus and deafness, right ear, May 24, 1957. Several hours later he had a disturbance of equilibrium of three hours' duration. The deafness and tinnitus has persisted. Between May and October, 1957, he had a disturbance of equilibrium following each flight.

Hearing Tests: The audiogram was essentially normal on June 21, 1954. A review of the audiograms accompanying the patient revealed a progressive hearing loss of the perceptive type, right ear, between June 11, 1957, and December, 1957.

AUDIOGRAM—December 4, 1957.

	250	500	1000	2000	3000	4000	6000	8000
Right Ear:	50	70	75	75	65	60	45	35
Left Ear:	0	0	0	5		5		–5

Speech reception threshold—Right Ear: 68 db. Left Ear: –3 db.

Discrimination score—Right Ear: 8 per cent. Left Ear: 96 per cent.

PGSR—Right Ear: 60 db, 70 db. Recruitment complete.

Caloric test—Right: Normal. Left: Normal.

Case 5. A.N.D., Civilian Employee, Age 43. On May 8, 1958, the pa-

tient developed roaring tinnitus and decreased hearing. Hearing loss and tinnitus were exaggerated by noise exposure. There was no history of vertigo.

Hearing Tests:

AUDIOGRAM—July 30, 1959.

	250	500	1000	2000	3000	4000	6000	8000
Right Ear:	45	55	45	45	50	45	35	20
Left Ear:	5	10	5	10	15	35	15	15

Speech reception threshold—Right Ear: 42 db. Left Ear: 5 db.

Discrimination score—Right Ear: 46 per cent. Left Ear: 68 per cent.

Diplacusis. Complete recruitment.

Caloric test—Right: Normal. Left: Normal.

Case 6. K.H.G. (The history and hearing tests were taken by Dr. Scott N. Reger, University Hospitals, Iowa City, Iowa, who kindly gave his permission to present them.) The patient had a normal audiogram in September, 1955. He awakened on the morning of May 1, 1956, with a feeling of fullness in the right ear. An audiogram revealed defective hearing of the perceptive type, right ear, more marked for the low frequencies than for the high. The hearing returned to normal over a three-day period. On February 6, 1958, the patient awakened with tinnitus, vertigo and noticeable hearing loss in the right ear. After the episode the hearing returned to normal. On March 19, 1958, he had a similar episode following which the hearing returned to normal. Between then and October, 1958, he had several more episodes. Since the episode on October 2, the hearing has not returned to normal.

Hearing Tests:

AUDIOGRAM—May 21, 1959.

	125	250	500	1000	2000	4000	8000
Right Ear:	25	25	30	30	35	40	70
Left Ear:	–5	–5	–5	5	10	5	10

Speech tests and caloric test were not available.

Patient referred to a physician for treatment.

Case 7. W.W.H., Staff Sergeant, Medical Technician, Age 29. In January, 1950, the patient had vertigo and nausea for five hours. No tinnitus or deafness at the time. He has been aware of defective hearing in his right ear since 1954. On February 11, 1959, he had a recurrence of vertigo and nausea. His hearing fluctuates.

Hearing Tests: Audiograms between November 22, 1954, and February 12, 1959, reveal fluctuating hearing, right ear. Defective hearing when present is of the perceptive type.

AUDIOGRAM—February 12, 1959.

	250	500	1000	2000	3000	4000	6000	8000
Right Ear:	35	35	55	45	50	50	35	15
Left Ear:	–10	–5	–5	45	50	55	35	10

Speech reception threshold—Right Ear: 50 db. Left Ear: –5 db.

Discrimination score—Right Ear: 28 per cent. Left Ear: 68 per cent.

Caloric test—Right: No reaction. Left: Normal.

SISI:

Right Ear:	0	0	60%	100%	100%	100%	20%	
Left Ear:	0	0	0	0	90%	95%	35%	

Békésy pure tone threshold tracings:

Right Ear: From 100 cps to 750 cps the continuous tracing was the same as the interrupted tracing. From 750 cps to 10,000 cps the continuous tracing drops 10 db from the interrupted tracing.

Left Ear: From 100 cps to 2,500 cps the continuous tracing was the same as the interrupted tracing. From 2,500 to 10,000 the continuous tracing drops from 7 to 23 db below the interrupted tracing.

*Case 8.** T.C.H., 1/Lt., Navigator, Age 26. A diagnosis of Ménière's disease was made on this patient April 2, 1958, by Dr. Fitzgerald, London, England. At the time the patient had a perceptive hearing loss in his right ear accompanied by disturbance in equilibrium. The record of this consultation was not available. There has been no recurrence.

Hearing Tests:

AUDIOGRAM—October 12, 1959.

	250	500	1000	2000	3000	4000	6000	8000
Right Ear:	–10	–10	–10	–5	–5	–5	10	0
Left Ear:	–10	–10	–10	–10	–10	–10	5	–10

Speech reception threshold—Right Ear: 0. Left Ear: –2 db.

Discrimination score—Right Ear: 70 per cent. Left Ear: 80 per cent.

Caloric test—Right: Normal. Left: Normal.

Case 9. H.J.L., Lt. Colonel, Pilot, Age 42. In February, 1959, the patient noted sudden drop in hearing, left ear, during a one-day period, not associated with tinnitus or vertigo. Denied history of vertigo.

Hearing Tests:

AUDIOGRAM—December 9, 1959.

	250	500	1000	2000	3000	4000	6000	8000
Right Ear:	–10	–10	–10	0	–5	55	40	60
Left Ear:	30	35	40	50	60	75 (no response)		

Speech reception threshold—Right Ear: 6 db. Left Ear: 52 db.

Discrimination score—Right Ear: 90 per cent. Left Ear: 48 per cent.

Recruitment is complete.

Békésy audiograms—typical of cochlear lesion on left.

SISI:

Right Ear:	–	–	5%	–	–	–	–	–
Left Ear:	100%	–	95%	–	–	100%	–	–

Caloric test—Right: Normal. Left: Normal.

Case 10. W.K.R., Major, Pilot, Age 40. In 1949, the patient developed tinnitus in the left ear. The hearing on the left fluctuates. The first

*This case is included because the author has confidence in Dr. Fitzgerald's good judgment and, secondly, because his consultation was reviewed by several headquarters, including the Office of the Surgeon General, USAF.

episode of vertigo occurred in 1951, and there have been four episodes since.

Hearing Tests: The audiograms in the patient's records reveal fluctuating hearing, left ear. The hearing defect is of the perceptive type.

AUDIOGRAM—July 3, 1957.

	250	500	1000	2000	3000	4000	6000	8000
Right Ear:	–5	–5	–10	–10	–5	–5		–10
Left Ear:	55	55	60	50	35	30	30	30

Speech reception threshold—Right Ear: 9 db. Left Ear: 55 db.

Discrimination score—Right Ear: 86 per cent. Left Ear: 14 per cent.

Recruitment present.

Caloric test—Right: Normal. Left: No reaction.

Case 11. J.K.S., Captain, Senior Observer, Age 36. In December, 1952, patient noted buzzing in his right ear and in November, 1956, he developed disturbance of equilibrium. He had nausea and vomiting of 15 minutes' duration. About this time he noted a progressive hearing loss on the right. There was a recurrence in January, 1957.

Hearing Tests:

AUDIOGRAM—August 13, 1957.

	250	500	1000	2000	3000	4000	6000	8000
Right Ear:	35	40	45	30	30	40	75	65
Left Ear:	0	0	15	15	10	15	30	25

Speech reception threshold—Right Ear: 39 db. Left Ear: 6 db.

Discrimination score—Right Ear: 32 per cent. Left Ear: Was not tested.

Complete recruitment.

Caloric test—Right: Markedly hypoactive. Left: Normal.

Case 12. D.P.S., 1/Lt., Instructor Pilot, Age 27. On April 14, 1958, the patient developed roaring and deafness in his right ear. Audiogram made at the time showed hearing loss in the right ear, especially for the lower frequencies. Later that day he developed vertigo.

Hearing Tests: The patient had a normal audiogram on February 11, 1958, on June 9, 1958, and in May, 1960.

AUDIOGRAM—April 14, 1958.

	250	500	1000	2000	3000	4000	6000	8000
Right Ear:	30	25	15	–5	15	25	15	
Left Ear:	0	0	0	0	5	10	20	5

Speech reception threshold—not tested.

Discrimination score—not tested.

Case 13. R.J.W., Captain, Senior Pilot, Age 35. The patient noted defective hearing in 1954. The hearing loss became suddenly worse and was accompanied by a buzzing tinnitus in November, 1955. Four days later he developed vertigo which lasted two hours. The vertigo recurred every three to four days. Following the vertigo the hearing returned to "normal." The patient had an episode of vertigo on December 13, 1956, during consultation. The hearing did not return to normal following this episode.

Hearing Tests:

AUDIOGRAMS—December 12, 1956.

	250	500	1000	2000	3000	4000	6000	8000
Right Ear:	10	10	5	15	35	40	65	35
Left Ear:	–10	–10	–10	–10	15	15	25	5

Speech reception threshold—Right Ear: 4 db. Left Ear: –6 db.

Discrimination score—Right Ear: 70 per cent. Left Ear: 68 per cent.

December 13, 1956.

Right Ear:	35	40	40	25	35	40	40	50

Speech reception threshold—Right Ear: 38 db.

Discrimination score—Right Ear: 50 per cent.

August 26, 1957.

Audiogram and speech tests similar to December 13.

Case 14. H.D.W., Staff Sergeant, Mechanic, Age 26. The patient has been aware of hearing loss on the right since early 1957. Since April, 1957, he has had recurring episodes of vertigo and disturbance of equilibrium.

Hearing Tests:

AUDIOGRAM—April 25, 1960.

	250	500	1000	2000	3000	4000	6000	8000
Right Ear:	55	70	70	60	65	65	50	55
Left Ear:	5	0	–5	5	20	20	25	35

Speech reception threshold—Right Ear: 65 db. Left Ear: 4 db.

Discrimination score—Right Ear: 30 per cent. Left Ear: 80 per cent.

Recruitment present.

SISI:

Right Ear: 90 to 100 per cent at all frequencies.

Left Ear: 5 per cent up to 1000 cps; 25 to 50 per cent through 4000.

Caloric test—Right: Normal. Left: Normal.

DISCUSSION.

Audiologic findings.

Perceptive deafness was diagnosed on the basis of an elevated threshold for air and bone conduction on the pure tone audiogram for the following cases: 1, 4, 5, 6, 7, 8, 9, 10, 11, 13, and 14. Case 8 is included because of the reputation of the consulting physician, even though his consultation report was not available. Audiometric curves typical of hydrops of the labyrinth—that is, with a greater hearing loss for low tones than for high tones—were found in cases 1, 2, 3, 5, 6,

10, and 12. Fluctuating hearing was noted in cases 1, 2, 3, 6, 7, 8, 10, 12, and 13. Fluctuating hearing is spontaneous and independent of treatment. Recruitment was tested by the alternate binaural method of Fowler. It was found to be present in cases 1, 4, 5, 9, 10, 11, 13, and 14. Diplacusis was infrequently looked for. It was found in cases 1 and 5. The discrimination score was obtained using the Rush Hughes recordings. Normal scores in our laboratory range between 75 and 85 per cent. Scores between 8 per cent and 52 per cent were found in cases 1, 4, 5, 9, 10, 11, 13, and 14. Scores within the normal range were obtained in cases 2, 3, and 8. The pure tone audiograms were within normal limits at the same time. The test results were not available on case 6. The test was not performed on case 12. The difference limen for intensity (SISI) was obtained on cases 9 and 14. In both cases the results indicated a cochlear-type lesion. A continuous and interrupted sweep frequency Békésy audiogram was obtained on cases 9 and 14. In both cases the results demonstrated a cochlear-type lesion.

Chronology of appearance of symptoms.

Vertigo appeared as the first and only symptom in only one case—No. 7. Vertigo and deafness occurred simultaneously in two cases—Nos. 3 and 8. In the other 11 cases, deafness alone, tinnitus alone or deafness and tinnitus appeared from minutes to as long as four years before vertigo appeared. In two instances vertigo has not been documented as of the present—cases Nos. 5 and 9. Case 9 was a pilot and had been examined by several physicians previously. He was aware that a diagnosis of hydrops of the labyrinth would probably result in his removal from flying. He may have elected to deny this symptom to the examining physician although he inadvertently indicated to the audiologist that he had had vertigo. The evidence for a diagnosis of hydrops on case 12 is admittedly flimsy; however, the air conduction audiogram of April 14 shows the typical curve of hydrops for the right ear. In addition, the right labyrinth continued to be hypoactive two years after the original episode.

Tests of labyrinthine function.

In all instances the ear canal was filled with ice water which remained in the canal for 30 seconds. Case 1 showed hyperactivity in the healthy ear and a normal reaction in the diseased ear. The results of the test were not available on case 6. No vestibular reaction of the right ear could be obtained on cases 7 and 10, even by douching with ice water. Cases 11 and 12 showed a hypoactive response in the involved ear. Appropriate diagnostic steps were taken to rule out acoustic neuroma in cases 7, 10, 11, and 12.

AVIATION MEDICINE IMPLICATIONS.

The Flight Surgeon should perform an audiogram on an aircrew member whenever he reports tinnitus with or without deafness and/or vertigo, because certain audiologic findings are characteristic of hydrops. A thorough audiologic examination should be performed when a unilateral perceptive hearing defect is found. The fact that the hearing may fluctuate should be kept in mind. Many patients with spontaneous tinnitus or tinnitus induced by acoustic trauma will be examined for each one who has or will develop hydrops of the labyrinth. The possible occurrence and significance of vertigo should not be discussed with the patient. When the audiologic findings are conclusive (*e.g.*, cases 5 and 9) even though vertigo has not occurred, the author believes that the patient should no longer remain on flying status, since it is impossible to predict if, when and where an episode of vertigo may occur. There is no question that flying safety would be compromised if an aircrew member had an episode of vertigo while flying.

Naturally, the vertigo of hydrops should not be confused with aviator's vertigo or disorientation. The fact that McNally and Stuart[4] reclassified 24 out of 78 cases should not be interpreted as meaning that one out of three aircrew members will be removed from flying unnecessarily. A careful review of their report reveals that only one (case 57) of the 78 had a diagnosis, after re-evaluation, which would possibly not be considered disqualifying for flying.

The method of treatment has been omitted intentionally.

It is beyond the scope of this paper. Most of the patients were free from vertigo at the time of examination.

SUMMARY.

While no definite conclusions can be drawn from the above case presentations, the following facts concerning hydrops of the labyrinth were clearly demonstrated:

1. The hearing defect due to hydrops of the labyrinth can be accurately differentiated from other types of hearing defects.

2. Defective hearing is frequently observed before vertigo occurs.

3. The early diagnosis is of particular importance to Aviation Medicine.

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THE OPEN REDUCTION OF NASAL FRACTURES.*

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St. Paul, Minn.

The discussion herein relates to the realignment and fixation of certain fractures of the nasomaxillary complex and nasal septum by methods more direct than the usual closed manipulative procedure. These fractures may be of the bony pyramid or of the cartilaginous septum and nasal projection. They constitute a limited per cent of the total of nasal fractures. The open techniques are presented because superior results can be obtained by one or a combination of them as compared to traditional closed methods in:

1. Extensive comminution of nasomaxillary and mesoethmoid bones with or without involvement of lacrimal bones, ethmoid labyrinth or vomer bone.

2. Fracture separation at the nasion.

3. Union in mal-position of recent (2-8 weeks) nasomaxillary fractures.

4. Immediate (0-2 weeks) nasomaxillary fractures which defy anatomic realignment by closed manipulation.

5. Fracture of the cartilaginous septum and nasal projection.

COMMINUTED FRACTURE OF THE NASOMAXILLARY COMPLEX AND MESOETHMOID.

Eggshell comminution of the bony nasal projection (see Fig. 1) requires a maintenance force to retain protrusion and a properly narrowed base. Protrusion can be provided by suspension apparatus from a head cap but at the cost of a wide base deformity. Both projection and a narrow base can

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be established and held by transfixion wires tied over malleable lateral plates. Two wires (32 gauge steel) are passed through the nasomaxillary base—one at the caudal end and one more cephalad. The lines of comminution aid in the passage, which is effected by a strong straight cutting edge needle mounted in a hand drill. The needle is not passed until

Fig. 1. Eggshell comminution of the bony nasal projection.

the entire nasomaxillary mass is overelevated by an assistant, with rubber-covered artery forceps placed high in each nasal vault. Draped over each forceps is a sheet of polyethylene film (see Fig. 2) which will be retained in the vault by the transfixing wires until the shredded mucoperiosteum has relined the septo-nasal vault without adhesive obliteration. Malleable lead plate of the calibre used in grounding plates is cut with a heavy scissors into two ovals to fit the bony portion of the nasal projection. They are properly convexed to avoid skin pressure at the edge and are drilled for passage of the wires. When the wires are tied over the plates the latter force

the comminuted mass snugly into the elevated contour and assure the proper basal narrowing (see Fig. 3).

FRACTURE SEPARATION AT THE NASION.

Separation at the glabellar root usually occurs only with direct lacerating injury, as by a projecting edge (see Fig. 4). The soft tissue access to the skeletal injury is invaluable in the repair. The urge to close the soft tissue laceration should

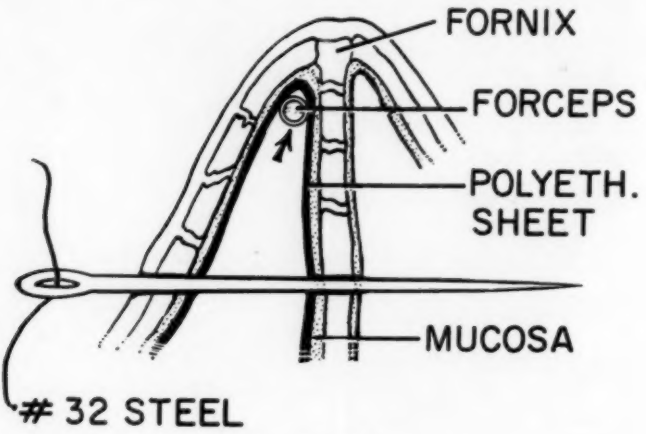

Fig. 2. Basal transfixion technique.

be subordinated to skeletal realignment. Major fragments are perforated by extremely fine drill points taking care the while to preserve periosteal attachments. Gauge 36 steel wire suture through the drill holes is used to tie the fragments into correct approximation and to close the nasal mass to the frontal root (see Fig. 5). Soft tissue closure from periosteum to skin is then readily accomplished without tension.

UNION IN MALPOSITION.

Unsatisfactory realignment of nasomaxillary fractures and failure to seek realignment create both a cosmetic and func-

Fig. 3. Retention of contour and projection with steel suture and malleable lead plates.

tional defect which becomes more glaringly apparent as the ecchymosis and swelling resorb. When the fracture fragments completely fuse in malalignment the smooth contour of the bony nasal components is permanently lost. The subsequent rhinoplastic procedure must include techniques to iron out the irregularities.

When the delay period of malunion has not allowed complete bone fusion the fracture components can be separated by intranasal osteotomy and manipulation. Complete freeing and mobility of the fragments are established before realign-

Fig. 4. Fracture separation at the nasion.

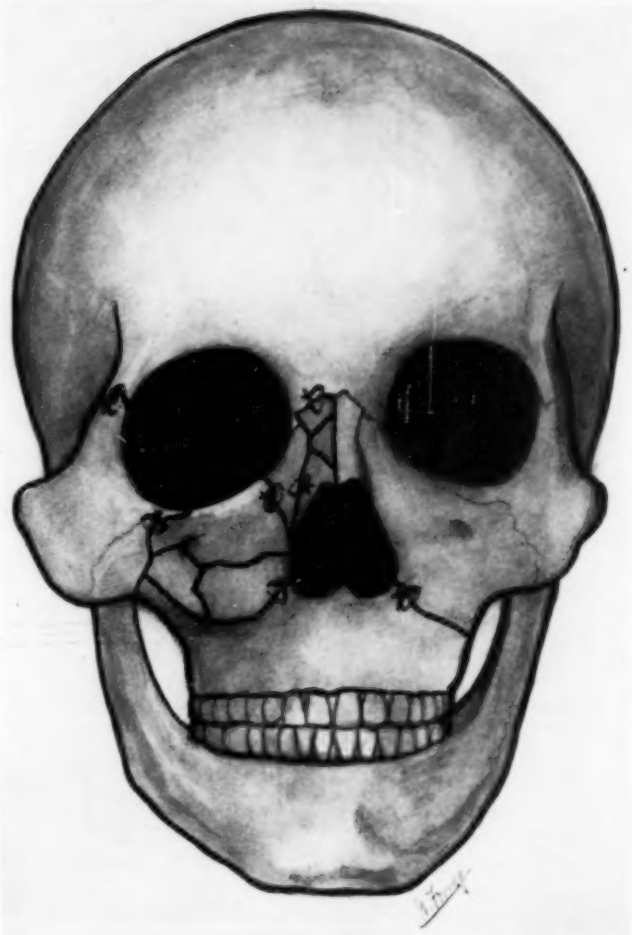

Fig. 5. Reapproximation and fixation by skeletal wire loops.

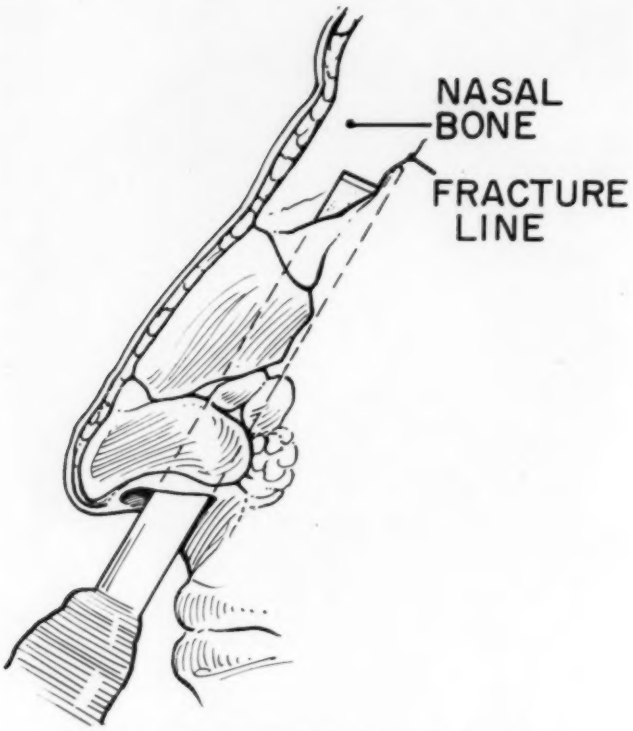

Fig. 6. Intranasal realignment with guarded osteotome.

ment. Osteotomy performed intranasally (see Fig. 6) violates only the mucosa which has already been torn by the fracture. Elevation of the soft parts from the nasal dorsum is not necessary so soft tissue reaction and thickening are minimized and organization is no problem. It is best to use a guard-tip osteotome so mischance will not allow it to slip through the fracture line and violate the skin.

UNSATISFACTORY REALIGNMENT BY CLOSED MANIPULATION.

Displacement of fracture fragments is occasionally only par-

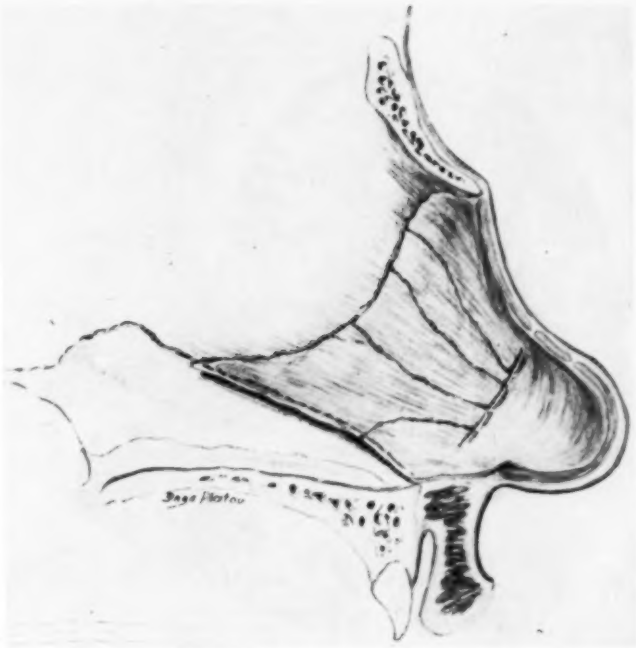

Fig. 7. Saddle deformity due to fragmentation of the cartilaginous septum. Note the relative "hump" formed by the caudal end of the nasal bones.

tially correctable by closed manipulation. This is usually due to a displaced fragment not accounted for in the general realignment. It usually is a fragment of the nasomaxillary process recessed medially to the maxillary fracture line, or to a fragment of mesoethmoid plate displaced and locked laterally to the remainder of the plate. The guard tipped osteotome used intranasally as a lever will sometimes facilitate repositioning by working through the mucosal laceration. External manipulation forces combine with the levering to remount the fragments. A gauze strip in the vault and an external moulded dressing are then used for maintenance.

Fig. 8. Hematoma support of the upper lateral cartilage contour conceals the injury. The soft spot in the dorsum caudal to tip of the nasal bones is diagnostic.

FRACTURE OF THE CARTILAGINOUS SEPTUM AND NASAL PROJECTION.

The residue of uncorrected fractures of the cartilaginous septum and nasal projection are both cosmetic and functional disability (see Fig. 7). Saddle deformity of the cartilaginous dorsum results in a relative hump at the tip of the nasal bones. Fragmentation and displacement of the cartilaginous septum causes a pinched tip, columellar retraction and nasal obstruction. The dissected mucoperichondrium of the vault of the nasal vestibule lowers the respiratory fornix permanently. The latter is not correctable by any rhinoplastic technique—not even an implant graft in the vault. The fracture of the

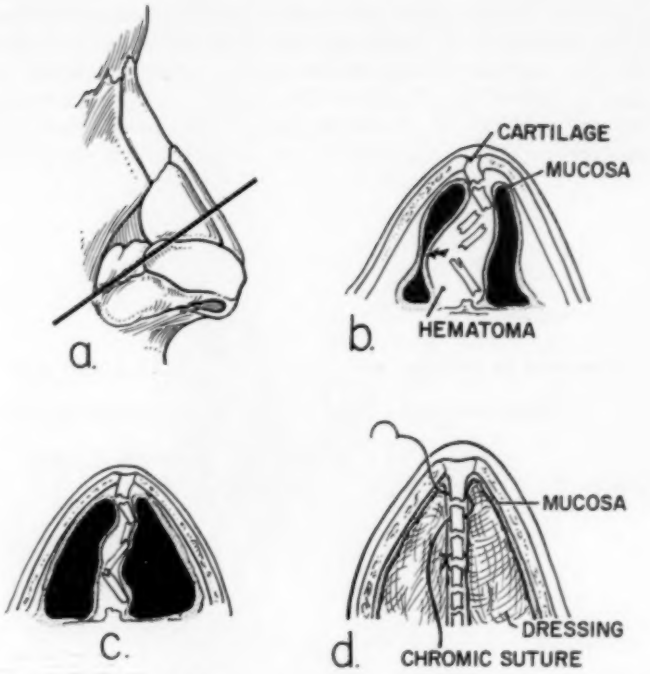

Fig. 9. a—Plane of section. b—Intranasal ballooning of the mucoperichondrial flaps and septal cartilage fragmentation. c—Organization and healing without open reduction and direct realignment. Permanent lowering of the respiratory vault. d—Suture of mucoperichondrial flaps and cartilage at each fracture line.

cartilaginous components does not show externally, primarily because the hematoma supports the upper lateral cartilages (see Fig. 8). Eight weeks after injury, when the hematoma has resorbed and the constrictive forces have pulled the upper lateral cartilages down to the gnarled septal fragments and the foreshortened mucosal vault, the saddle and relative hump externally become apparent. This can be diagnosed and predicted by pressing the examining finger down the dorsal nasal line. If a soft depression is evident immediately caudal to the tip of the nasal bones, serious cartilaginous septal injury has

occurred. Intranasally the septal mucoperichondrium will have ballooned with the hematoma to fill the nasal space from inferior turbinate to inferior turbinate (see Fig. 9 b). An open reduction is mandatory. The result of proper immediate surgery will be far better than later rhinoseptoplasty can produce.

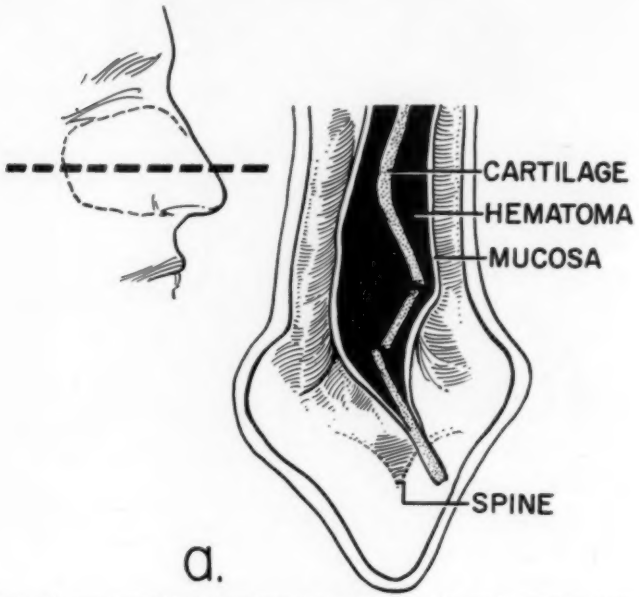

Fig. 10. a—"Accordion" fracture dislocation of the cartilaginous septum due to direct antro-posterior impact.

The surgery is not difficult for anyone familiar with intranasal techniques. Incision is made in one nasal vestibule as for a classic submucous resection. When the hematoma is evacuated the septal fragmentation presents an interesting disarray. One begins to understand the origin of the confusion and distortion of the commonplace post-traumatic anterior septum. Realignment is begun at the dorsal line and is supported by the first folds of the intranasal dressing strip. As the accordion of fragments is unfolded a mattress suture of

4-0 chromic suture is placed through and through mucosa and cartilage to embrace each fracture line (see Fig. 9 d). The inferior fragment is replaced on the maxillary ridge, and this is maintained by a basal suture passed through vestibular skin under the anterior nasal spine and then through mucosa and inferior cartilaginous fragment. The caudal end of the septum

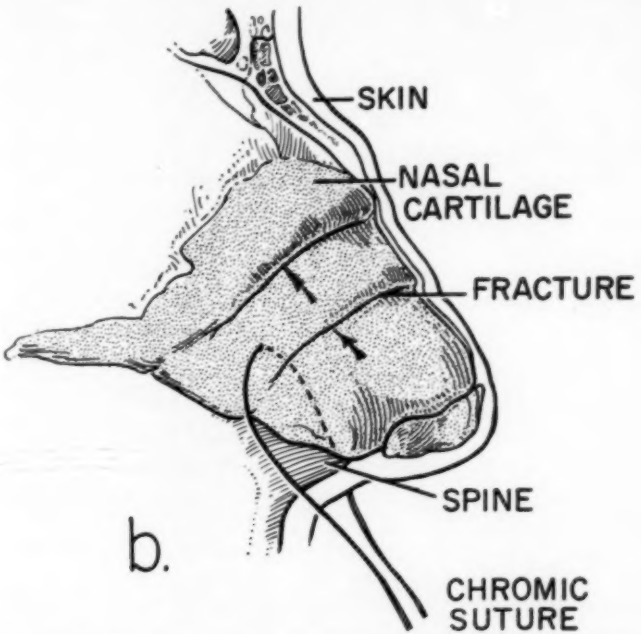

Fig. 10. b—Placement of a 3-0 chromic suture through mucoperichondrium and cartilage of fragment posterior to the anterior nasal spine.

is thus drawn forward into its anterior position and the normal columellar support is restored (see Fig. 10 c). The intranasal dressing is then completed, and is left in place for five days.

When the injury force impacts directly against the cartilaginous nasal tip and alveolar maxilla rather than down

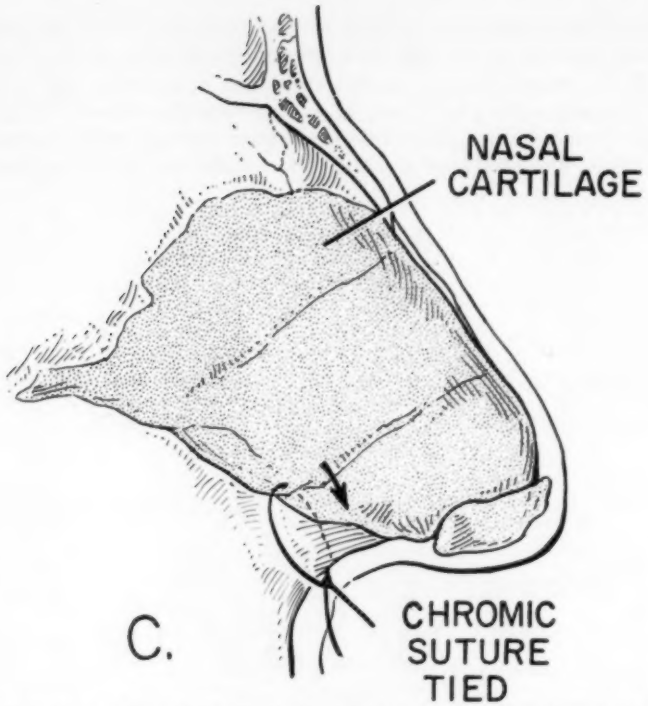

Fig. 10. c—Suture then passed through skin of vestibular floor, beneath anterior nasal spine and into opposite vestibule. The tie unfolds the posterior accordioning and maintains forward repositioning.

against the dorsum, the septum folds backward in accordion fashion with dislocation from the spine with or without its fracture (see Fig. 10 a). Usually a swollen and ecchymotic upper lip results also, and occasionally injury is done to the upper incisor teeth. Moderate swelling of the nasal tip may occur, and moderate septal edema may partially occlude the vestibule. The skin and perichondrium are very adherent to the septal cartilage anteriorly, and a ballooning hematoma does not always result.

The posterior folding and displacement of the septum are readily corrected by anterior traction with a toothed forceps.

When the septum is released it promptly recesses. A relatively simple and often satisfactory solution is that afforded by a 3-0 chromic suture placed through the mucosa and cartilage of the fragment posterior to the anterior nasal spine (see Fig. 10 b). When this suture is passed through vestibular skin, beneath the spine and out into the opposite vestibule and drawn up snugly, the forward traction straightens the whole accordion and maintains it while the perichondrium at the septal base refixes to the maxillary crest and the nasal spine (see Fig. 10 c).

SUMMARY.

A relatively small group of nasal fracture cases is best treated by open reduction methods.

These are defined and the techniques discussed and illustrated.

AMERICAN ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY.

The 1961-1962 Home Study Courses in the basic sciences related to ophthalmology and otolaryngology, which are offered as a part of the educational program of the American Academy of Ophthalmology and Otolaryngology, will begin on September 1 and continue for a period of ten months. Detailed information and application forms can be secured from Dr. William L. Benedict, executive secretary-treasurer of the Academy, 15 Second Street S. W., Rochester, Minnesota. Registrations should be completed before August 15.

PROLONGED BONE CONDUCTION: OBSERVATIONS ON MAN AND ANIMALS.*†

RALPH F. NAUNTON, M.D.,
and
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Chicago, Ill.

Eight years ago Hirsh[5] commented that "Cursory examination of the literature on bone conduction gives one the impression that the application of a tuning fork to the skull, tests only the sensitivity of the inner ear and auditory nervous systems. . . . This is clearly not the case." Today it is abundantly clear to otologists that structural or pathological changes in the outer or middle ears may and frequently do influence the measurements made in clinical bone conduction audiometry.

It is of interest to retrace the paths that our thinking has followed in connection with what was formerly described very literally as prolonged bone conduction. Schwabach in 1890 first drew attention to the fact that bone conduction could be better than normal in the presence of middle ear lesions, an observation that was confirmed 18 years later by Bezold.[2] In 1926 Politzer[9] again confirmed these observations, but also went so far as to provide a theoretical explanation which would stand up fairly well today in the light of our still inadequate understanding of bone conduction physiology. In the next 20 years Hallpike, Pohlman,[8] Barany,[1] and Bunch[3] each independently denied that there could be an absolute or real prolongation of bone conduction, and attributed the apparent increase of bone conduction hearing above normal to ambient noise. To quote Barany[1] writing in 1938, "The reason

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why Schwabach and his contemporaries found an increased bone conduction in middle ear deafness was the existence of an appreciable noise level in their testing rooms. In a noisy room the threshold of all hearing is limited by the noise level; thus, in such a room, an ear with a middle ear lesion having considerably decreased air-sound acuity but only slightly decreased bone-sound acuity, will be in a more favorable position for hearing by bone conduction than will a normal ear. While the latter *(that is the normal ear)* is disturbed by the noise, the deaf ear does not hear the noise, and its slight decrease of bone conduction is more than compensated for by its greater immunity against air-borne noise."

In 1952 Hirsh[5] further suggested that little change in low tone bone conduction is produced by lesions central to the cartilaginous meatus, adding that, in the majority of instances, enhanced bone conduction hearing is due to a *relative* improvement rather than to an *absolute* improvement in bone conduction. I believe it is fair to state that today we all agree with Schwabach, in stating that clinical bone conduction is prolonged or enhanced under some circumstances and, when we can give complete explanations based on experimental findings, it is probable that they will fairly closely resemble the explanation offered by Politzer 34 years ago.

Probably the most dramatic changes in low tone bone conduction hearing resulting from middle ear changes are seen in secretory otitis, in which the tympanic cavity behind an intact drum is fluid-filled. In order to demonstrate this effect to you, we sort some cases of *bilateral* secretory otitis in adults —bilateral because we wished to carry out accurate bone conduction tests without masking. We have only three such cases, but with each of them we carried out bone conduction tests in a sound treated room using a Békésy audiometer. Bone conduction was tested first while the pathological middle ear condition was at its height; then when the middle ear condition had subsided, we repeated the Békésy bone conduction tests. Our results demonstrated the now familiar phenomenon, noticed by Schwabach, of an absolute and above normal improvement of low tone bone conduction hearing while the mid-

dle ears were fluid-filled, the average improvement for the three cases being illustrated by the solid circles (see Fig. 1).

Three normal listeners were tested twice in a similar fashion, the interval between each normal listener's two tests being about the same as the time taken for the secretory otitis cases to recover; these tests on normal listeners served as an approximate indication of the repeatability of Békésy bone

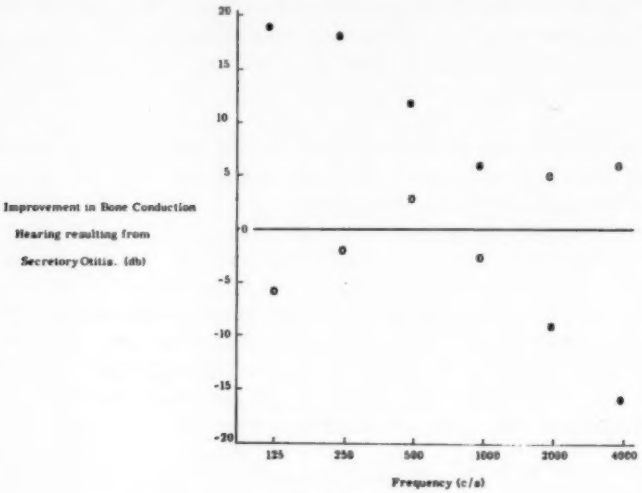

Fig. 1.

conduction tests, the average difference between first and second tests for the normals being shown on the same graph (open circles). We are justified in assuming that the change in bone conduction hearing demonstrated by these three cases is the result of the presence of a collection of fluid behind an intact drum.

Carhart,[4] in 1950, was the first to point out another and very familiar instance in which a middle ear lesion, this time otosclerosis, influences our measures of clinical bone conduction hearing; but in this instance, instead of a low tone improvement or threshold depression, the bone conduction

audiogram shows a characteristic threshold elevation, or bone conduction deterioration, affecting 1000 c/s through 4000 c/s, but greatest at 2000 c/s—in other words, the now classical Carhart notch.

Figures 2 and 3 show two good examples of the Carhart notch, both of these listeners being young otosclerotics.

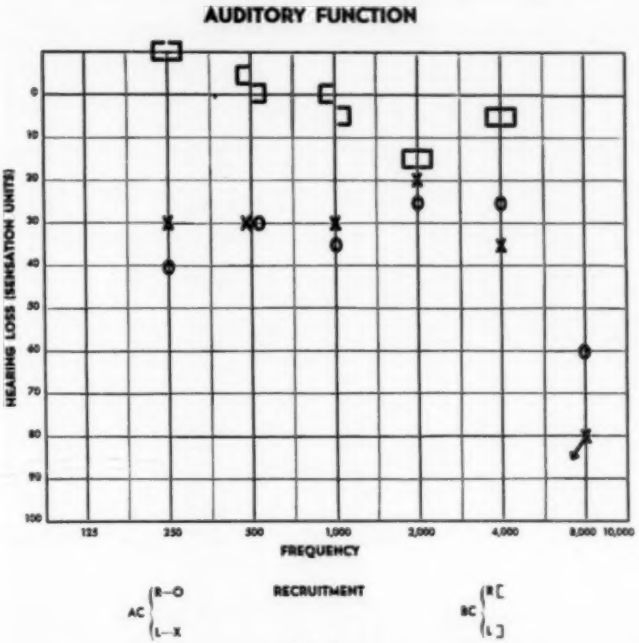

Fig. 2.

The Carhart notch occurs, although less commonly, in adhesive or suppurative conditions of the middle ear, but more frequently the bone conduction changes brought about by purely mechanical factors operating in the middle ear in these cases are above normal low tone bone conduction hearing and progressively worsened bone conduction hearing up to the limit of the audiometer. Lierle and Reger[7] drew attention to this in 1946.

Juers[6] (and later others) provided an interesting corroboration of Carhart's claim that the "notch" was the result of mechanical changes in the middle ear. When writing in 1948 he observed that the high tone bone conduction loss can at least partially be reversed or corrected by fenestration of the labyrinth. Now that, to quote the words we heard so often

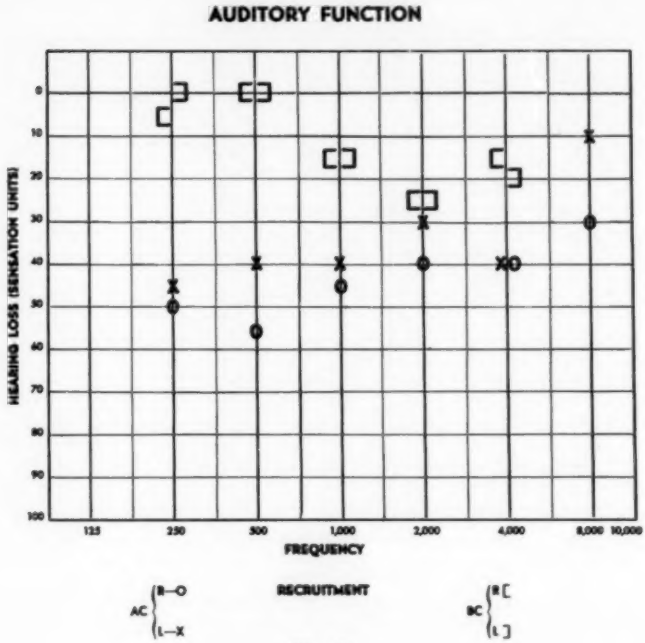

Fig. 3.

recently in Detroit, "we have buried the fenestration operation," we are seeing this same reversal of the Carhart notch following stapes surgery. Let me illustrate this point further by showing you the results of a case of mine, an otosclerotic in her 50's who was treated by stapedectomy and the insertion of a wire-Gelfoam prosthesis (see Fig. 4). The changes in bone conduction are of particular interest. We have had

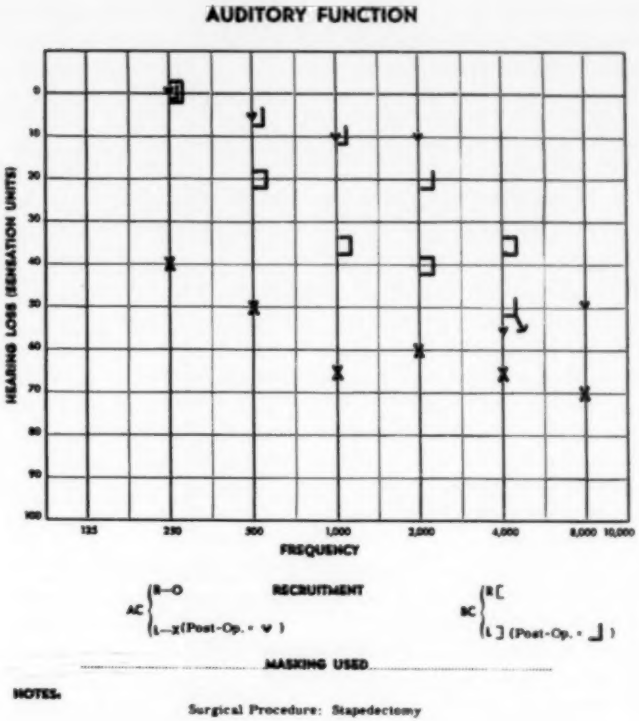

Fig. 4.

patients at the University of Chicago whose bone conduction hearing has returned more nearly to normal than this one, but this particular case is of considerable interest because the patient started off, before surgery, with an unusually severe Carhart notch.

From what little we know of the mechanism of bone conduction (and we still have no adequate explanation of the Carhart notch), it is not really surprising that surgery should reverse or correct the notch. It is, however, difficult to understand why, amongst a group of apparently similar otosclerotics, some will have the notch while others will not; and

why some cases show correction of the notch after surgery and others will show no changes in bone conduction following the same surgical procedure.

There is one very simple way in which we can experimentally change a normal listener's bone conduction hearing. It is by occluding, or covering, the ears. You are familiar with this, in an everyday sense, in that if you get water in one ear when you are in the shower, the loudness of your own

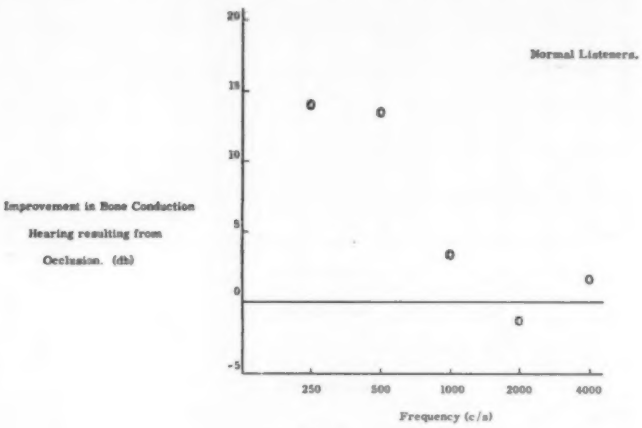

Fig. 5.

voice becomes much greater in the blocked or occluded ear. Bathroom baritones will be well aware of this and will also have noticed that the increased loudness is largely an increase of low tones. Covering or occluding the normal ear with a standard type of audiometric headphone gives much the same effect, but an effect that can more easily be measured.

Just as we were able to demonstrate the improvement of low tone bone conduction hearing that occurs in secretory otitis, so we can demonstrate the somewhat similar improvement that takes place when we cover or occlude the ear. To this end a group of 40 normal listeners was tested twice, bone conduction hearing being measured in a sound treated room,

first with the ears open and secondly while the ears were covered or occluded with standard audiometric headphones. Occluding the ears in this way produced an above-normal improvement in low tone bone conduction hearing, the group average improvement being shown (see Fig. 5). This change is described as the occlusion effect, and you will see at once that the improvement is somewhat similar, in degree and frequency distribution, to the effect observed in the secretory otitis cases; it must not, however, be assumed that mechanisms of the changes in the two groups are identical—they are closely related, but they are not identical. Notice that if we take some liberties with the figures on the ordinate the graph in Fig. 5 shows some uncanny resemblances to the bone conduction curve of an otosclerotic and his Carhart notch. Notice also in passing that we believe that when a middle ear is the sight of pathological changes it will often have prolonged bone conduction; but, whether or not its bone conduction in the open ear state is prolonged, covering the ear with a headphone will *not* produce an occlusion effect.

Dr. Fernandez and I have for some time been interested in attempting to understand some of these phenomena more clearly, and our first step has been to look for an objective demonstration of the occlusion effect, the change in bone conduction hearing that results when an ear is covered, in experimental animals. We used anesthetized guinea pigs for our experiments and recorded cochlear microphonics by means of round window electrodes. After exposure of each animal's middle ear and placement of the round window electrode, the middle ear cavity was closed again. A hearing aid type of bone conduction receiver was applied with pressure to the animal's skull in the median plane at the vertex. Pure tone stimuli were delivered to the bone conduction receiver and the magnitude of the resulting cochlear microphonics was recorded; thresholds were measured in terms of the least input required to produce a just-visible cochlear microphonic response on a cathode ray oscilloscope. The animals' ears were then occluded by placing a small ball of moist cotton at the opening of the cartilaginous meatus, and the measurements were repeated. Comparisons between the microphonic thres-

holds in the open ear and the occluded conditions gave us a measure of the change in bone conduction hearing resulting from occlusion in the experimental animal, the average changes shown by 12 animals being illustrated (see Fig. 6).

We were particularly interested in the frequency region below 2000 c/s, hence the many frequencies tested in this region. Note again the similarity between these changes, and the improvement in humans resulting from secretory otitis

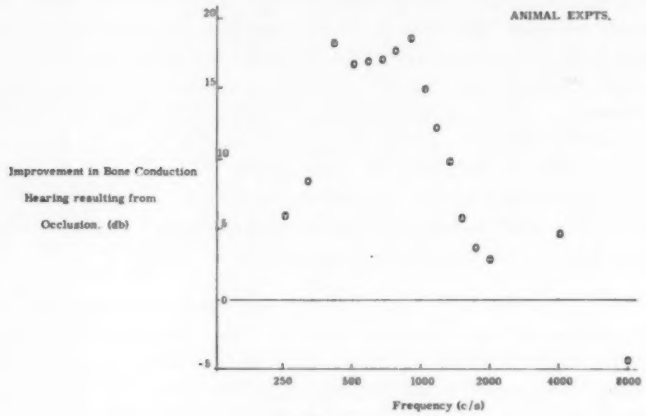

Fig. 6.

or from occluding the normal ear. The guinea pig shows bone conduction improvement at a slightly higher frequency, but the effects are very similar in magnitude and generally similar in frequency distribution, and once again we have something not too far removed from the Carhart notch.

The guinea pig experiments were controlled in several ways. For example, our tests were always repeated with the bone conduction receiver held a few millimeters clear of the skull in order to examine the effects of any air-borne sound radiated from the bone conduction receiver. Except at high frequencies, the air-borne sound was negligible; it is probable, however, that at 8000 c/s the stimulation was entirely via the air-borne route.

In addition to threshold changes, we were also able to record phase and other changes in the cochlear microphonics, but our investigations in this direction are incomplete and still continuing. More complete results will be published later.

I have spoken of the bone conduction changes that can readily be recognized in secretory otitis, otosclerosis, and when

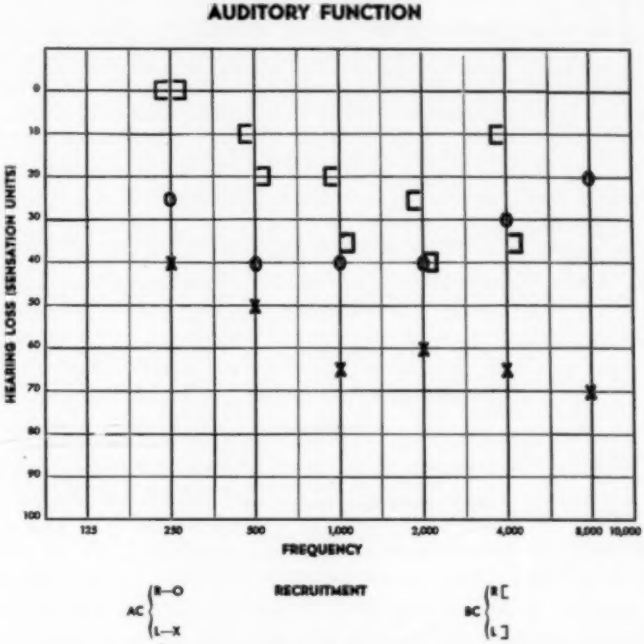

Fig. 7.

the normal human or animal ear is occluded. I have also illustrated the bone conduction changes we see after fenestration or stapedectomy. What is the significance of these phenomena we have been discussing? Foremost comes the general point we have been anxious to re-illustrate to you today—that it cannot be assumed that a bone conduction test is measuring

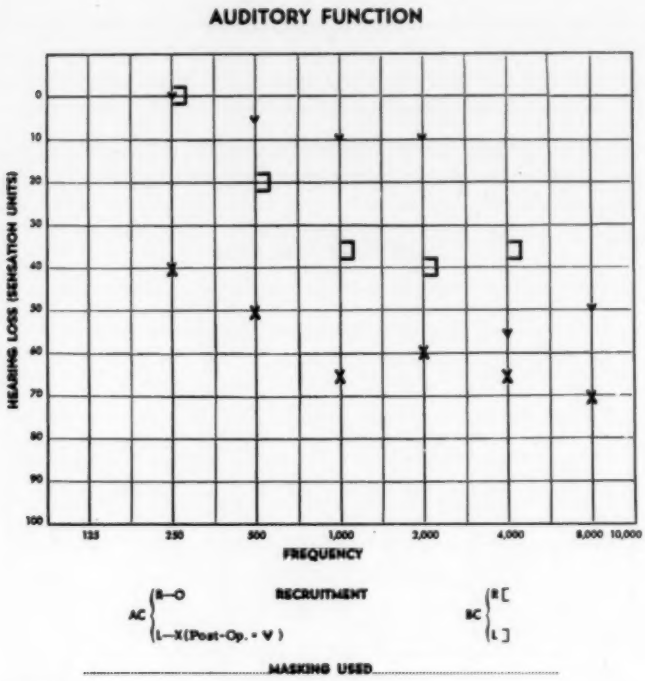

Fig. 8.

only cochlear reserve; cochlear reserve *is* being measured, but the results are always contaminated to a greater or lesser extent by the middle ear. To express this in another fashion we may say that our bone conduction norms are valid only so long as the middle ear is normal and as the tested ear is unoccluded. If we have reason to believe that a tested subject's middle ear is not normal we have to be on the look-out, as we interpret the bone conduction test results, for possible interference between middle ear pathology and bone conduction

threshold measurements. We have referred to some of the forms this interference may take; there may be others of which we are unaware at present.

As a further illustration let me return to the otosclerotic I mentioned earlier who had the striking Carhart notch. Figure 7 shows her pre-operative audiogram again. This lady was in her 50's and surgery could not be contemplated on her better ear; but if we were to operate on the poorer ear, the left, and gain 100 per cent closure of the air-bone gap, I would be giving her an ear only a shade better than the right ear even if we apply the Shambaugh correction. Fortunately, her left ear was explored with the very satisfactory results shown (see Fig. 8). The reasons for this were first the high PB score (96 per cent) which *suggested* that the elevated bone conduction thresholds did not indicate a sensorineural loss but a rather severe Carhart notch; moreover, if the bone conduction changes were due to true sensorineural changes, bone conduction at 4000 c/s would be far worse than was actually the case; the patient's willingness, after she had heard a complete explanation of our problems, to take a chance on getting a worth-while result from a near useless ear, was the final point that led to the decision to operate.

I mentioned earlier that another point of significance in this curious but important relation between bone conduction tests and the state of the ear's air conduction mechanism is that, if we wish to conduct tests with the tested ear occluded or covered by an air conduction receiver, we must use a different normal reference level that takes into account the occlusion effect and the change in bone conduction thresholds it entails. We must, therefore, be very cautious about adopting the currently popular method of clinical bone conduction testing that involves covering the tested ear with a headphone.

I have also mentioned that the patient with an abnormal middle ear, whether he does or does not show prolonged bone conduction when tested in the orthodox manner, will not show an occlusion effect when his ears are covered with headphones. If, therefore, we follow the recommendations of Rainville,[10] and more recently Jerger and Tillman,[11] and cover the ears

during all bone conduction tests, cases like the three cases of Secretory Otitis will have normal instead of "Prolonged" bone conduction at low frequencies; and many of our otosclerotics and cases with other varieties of middle ear damage will appear to have mild low tone nerve deafness.

It is clearly incorrect to say that it doesn't matter whether bone conduction is tested with open or covered ears. Certainly it matters to the otologist, who reads the audiogram; otologists expect to gain information of differential diagnostic value *as well as* measures of cochlear reserve from bone conduction audiograms (*e.g.*, prolonged B-C in secretory otitis).

By all means let us put the bone conduction receiver on the forehead instead of the mastoid; but let us not discard information of differential diagnostic value by covering up the ears and making bone conduction tests even more confusing than at present.

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THE NEW MEATAL PEDICLE GRAFT FOR RESTORATION OF THE EARDRUM.*

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A review of the European and American literature reveals a remarkable abundance of procedures for grafting tympanic membranes in the surgical reconstruction of the tympanic cavity. Whether this procedure is employed in myringoplasty (transmeatal tympanoplasty), Modified Type I tympanoplasty with exploration of the mastoid antrum and attic contents with preservation of the posterior bony canal wall, or in a Type II, III, IV or V tympanoplasty according to the Wullstein classification, is of little concern in this discussion since we are all aware of the variable degrees of intratympanic destruction which may direct one's approach in the repair of the sound conduction mechanism. In this paper we are primarily concerned with a method of skin grafting.

In 1953, Zollner[1] and Wullstein[2] of Germany published their work using free grafts in surgical reconstruction of the middle ear cavity. Following the experience of Ombredanne[3] in which he utilized a thin free graft to dry up a radical mastoid cavity and noticed an excellent hearing gain, Zollner then followed the same procedure, and found that the graft, if placed in apposition to the head of the stapes, served as an excellent substitute for the membrana tympani. He further noted that where wider areas were grafted, the thin full thickness graft seemed to hold up better.

In the same year Wullstein reported on 100 cases of tympanoplasty and obtained best results with a thin full thickness graft approximately 1 mm. in thickness.

In 1957, the author[4] reported on a series of tympanoplasty procedures performed over a three-year-period in which re-

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sults were summarized. In these procedures a thick split thickness graft was prepared from the postauricular area, at least 1 mm. in depth. At best, only 50-60 per cent of free grafts survived even though optimum precautions were observed, and prerequisites were ideal. It was observed that thick split thickness grafts were used with good results only when,

1. Using utmost care in preparing a large bed completely free of islands of epithelium to receive the graft.

2. The graft is uniform and at least 1 mm. thick.

3. The defect to be bridged is not too large, in which cases the promontory of the cochlea is used as a contact.

4. The free graft is in good contact with the "bed"; there is no overlapping of edges and the margins are in good apposition to the meatal epithelium. It was also noted that pedicle grafts were preferred to free grafts where possible.

For a number of years, pedicle skin flaps of various types have been used from time to time to close defects in tympanic membranes or to construct substitute tympanic membranes in cases of congenital meatal atresia. These pedicle flaps were made sometimes from the skin of the meatus but more often were made from the skin above or behind the auricle. These were rather awkward and difficult to swing into position and also left a rather pronounced scarring. Sourdille[5] also used a type of tympanomeatal flap that proved both convenient and practical for the fenestration operation; however, this pedicle flap was attached to the drum at twelve o'clock. This is the type of flap that Shambaugh and others in this country have used for the fenestration procedure and one which I had employed for fenestrations since my training with Doctor Shambaugh in 1950. Moritz,[6] prior to the published works of Wullstein and Zollner, maintained that pedicle grafts were necessary for tympanoplasty. Sooy[7] prefers a sliding pedicle graft made from the skin of the posterior meatal wall to close large marginal tympanic membrane perforations. Zollner and Wright[8] prefer to use the thick Thiersch split thickness graft which actually is a thin full thickness graft for simple myr-

ingoplasty. I think it can be generally said, however, that all other otologists at the present time are using Wullstein's full thickness graft taken from behind the ear.

Today, then, we still find a multiplicity of techniques employed in restoring the integrity of the eardrum, either in part or *in toto*, hence indicating that perhaps results from the methods employed in the hands of all otologists are not ideal. There are variations as to the donor site, variations in cutting, trimming and applying the graft. Some surgeons prefer gelfoam as a support; some prefer none, while others still prefer to contact the graft with the cochlear promontory where complete replacement of the drum is attempted. I am sure that all of these procedures are good in the hands of the surgeon who has developed his individual technique, but unless this procedure is simple and effective enough for all otologists to employ, it is not the ideal procedure to use.

Variations in technique and special precautions observed in myringoplasty or tympanoplasty procedures have evolved out of consideration to the following problems:

1. Maintenance or establishment of a good blood supply to the graft.

2. Adequate coverage of the drum defect or entire annular area if necessary.

3. Secondary sloughing and desquamation and adequate vibratory capacity.

Wright and Guilford[9] have emphasized the necessity of grafts of sufficient thickness to include the subepithelial capillaries since these are the vessels which establish contact with the grafted site.

William House[10] and Harold Schuknecht[11] advocated the use of the anterior portion of the external auditory canal for small drum defects. Schuknecht prefers free grafts from the postauricular area for large drum defects.

Shea,[12] Tabb[13] and others have used veins for repair of the drum. For the past two years, we have employed a meatal

pedicle graft technique which can be used regardless of size of defect or type of tympanoplasty performed, and which is quite different from any described in the literature. The author reported on this technique in less detail in an article presented at The Southern Medical Association Meeting in Atlanta in November 1959. To date, we have performed over 100 operations of all types using the pedicle meatal graft and find it much more gratifying in effecting good over-all results in all types of tympanoplasty.

ANATOMY OF THE MEMBRANOUS CANAL.

The skin overlying the bony canal wall is extremely thin and, of course, is too thin for grafting drum defects. Superiorly, at twelve o'clock, however, the cutis portion of the ear canal is thicker and covers some 50 per cent of the bony canal roof. Posteriorly, there is a sizeable amount of thick skin superficial to the bony canal, and anteriorly there is an abundant supply of thick skin supported by cartilage. Since sizes of ear canals are variable, the size of the skin pedicle varies, but most have measured at 10-14 mm. by 18-22 mm.

PROCEDURE.

It is important to note that an endaural incision is employed in all types of tympanoplasty or myringoplasty for several reasons:

1. It is almost impossible to prepare a pedicle flap large enough without the endaural incision.

2. It is most important to inspect the middle ear cavity and attic contents for cholesteatoma by osteotomy of the attic wall at the posterosuperior annular rim; or, one may want to inspect the mastoid antrum via tympanoatticotomy approach. Usually when one performs a Type I tympanoplasty or myringoplasty, the former procedure suffices and tympanoatticotomy is not found necessary.

3. The endaural incision for simple myringoplasty affords a great deal more exposure.

Routine incisions #1 and #2 are then employed as in any

endaural procedure with the exception that incision #1 does not extend as high along the preauricular fold and incision #2 is approximately 5 mm. external to the bony meatus. Incision #3 extends from the annular rim at four to five o'clock on the clock dial, is carried superficially to the external meatus and curves around anteriorly and superiorly to meet incision #2 at twelve o'clock. Posteriorly, the skin is thinned by removal of subcutaneous tissue and is elevated from the bony canal wall as far as the drum membrane. Care is taken to prevent tearing as the tympanomastoid suture line is encountered. Anteriorly, the skin is separated from subcutaneous tissue and particularly cartilage, with a Bard-Parker knife.

Utmost care is necessary in separating the skin from the petrotympanic suture line as it is much more pronounced than the posterior bony union. Elevation of the entire membranous canal is then relatively easy and is performed for the entire 360 degrees at the annular rim of the drum. Incision #4 is the annular incision and is performed with a right-angled elevator especially adapted for this incision. Incision #5 is a small incision posteriorly at eight o'clock on the clock dial which both lengthens the pedicle and facilitates rotation of the graft. The graft, now consisting of the entire meatal skin, and separated from the drum remnant by the annular incision, is intact and is attached to the pedicle posteroinferiorly as we note in Diagram 4. The pedicle is then elevated and the entire graft is tucked out of the operative field externally. Thus far, this entire procedure requires approximately 20-25 minutes.

Visibility of the drum area is greatly enhanced in this procedure, and denudation of the drum remnant and particularly the anterior bony wall concavity is afforded with great deal more facility. Inspection of the middle ear and attic contents is then made routinely following osteotomy of the attic wall.

Tympanoatticotomy is performed if deemed prudent, to rule out antrum disease. Intratympanic disease processes and dehiscence or ankylosis of the sound conduction mechanisms are not within the realm of discussion in this paper, but it

suffices to say that the pedicle graft may be used in any type of tympanoplasty procedure with good results. It may be used to replace all or any part of the drum.

In placing the graft, it will be noted the thicker posterior and superior portion of skin canal is placed over the annular area. No gelfoam is used, unless the entire drum is absent, in

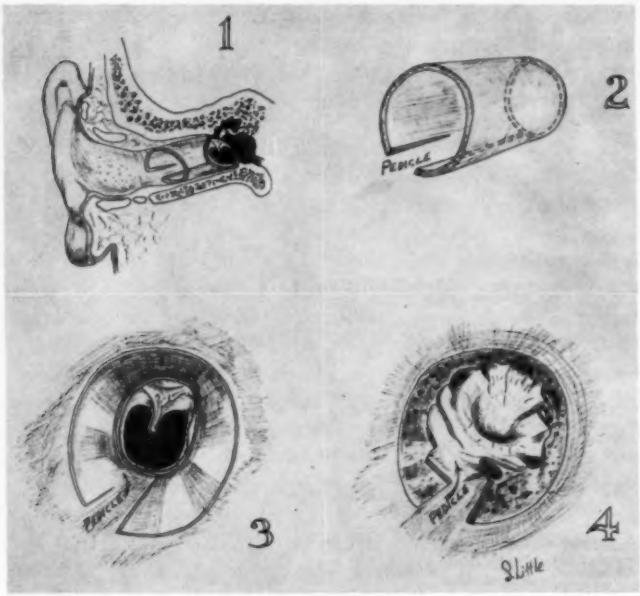

Fig. 1.

which case a small pledget is placed in the hypotympanum. It is most important in placing the graft to make certain the graft is contacting all parts of the drum remnant, particularly the anterior drum remnant or the annular rim as the case may be. A small piece of surgical rayon bathed in Polysporin Ointment is placed over the graft, and a tiny pledget of sea sponge is fit snugly in the graft pocket. This allows for elasticity and does not exert excess pressure on the graft. Many

times a thin full thickness graft is applied to the posterior bony canal wall to facilitate epithelization, but even without this, epithelization is usually rapid and complete.

Suture lines should be smoothed down even with the bony wall to facilitate epithelization and prevent polypoid growth formation. If the canal is a small one, it is well to widen it with a burr under irrigation to prevent postoperative narrowing. The packing is removed in twelve days. Tubal inflation is instituted in twenty-one days.

ADVANTAGES OF THE PEDICLE MEATAL GRAFT.

1. The incidence of take is much higher than that of free graft and the incidence to secondary breakdown much lower.

2. There is assurance of a definite blood supply to the graft. Whether the small pedicle plays a major role in supplying the blood to the graft, I do not know, as this has always been a debatable question since "fenestration days." I do know, however, that with the pedicle graft, satisfactory "take" is almost 100 per cent.

3. Since meatal skin is more like tympanic membrane skin than any other, there is very little desquamation of the graft following "take" and follow-up care is at a minimum.

4. The graft is easy to prepare, may be placed well out of the operative field, and epithelization of the denuded canal wall is usually rapid and complete.

5. There is greater exposure of the drum area.

6. It is a routine step by step procedure which can be performed by all otologists eliminating guesswork and is employed in all types of tympanoplasty.

RESULTS.

Eighty-one tympanoplasty procedures performed in an eighteen month period have been evaluated in which the pedicle meatal graft was utilized. Of these, thirty-seven operations were Type I or Type M-I. In all of these, either the attic or the mastoid antrum or both were inspected for pathological

changes. For example, all Type I tympanoplasties were accompanied with elevation of the drum posterosuperiorly, osteotomy at the posterosuperior bony rim and partial removal of attic wall if necessary for thorough examination of the ossicular chain and for mobility of the stapes. All M-I (Modified I) tympanoplasties were accompanied with tympanoatticotomy for inspection of the mastoid antrum, in addition to the above procedure.

On reviewing the statistics, it is noted that in Type I and M-I tympanoplasties, there was adequate "take" in 89 per cent of cases. Ninety-four per cent of these were still intact six to eighteen months following operation. In Type II, III, IV and V tympanoplasties, only 70 per cent of grafts were intact six to eighteen months following surgery. Failure in these cases was due largely to tubal pathology toward which allergic Eustachian salpingitis was a large contributing factor.

Total 81 Patients.

Type I Procedures	25
Good "Takes"	23
Secondary Breakdowns	2
Breakdowns Due to Otitis Media	2
Intact Drums—6 Months	21

Fig. 2.

Type M-I Procedures	12
Good "Takes"	12
Breakdowns	0
Intact Drums—6 Months	12

Fig. 3.

Type II, III, IV, V	44
"Takes"	31
Intact Drums—6 Months	31

Fig. 4.

Intact Grafts	6 Months
Type I	89%
Type M-I	89%
Type I, II, III, IV	70%

Fig. 5.

SUMMARY.

The author presents a method of grafting technique utilizing a pedicle meatal graft which is easy to perform, produces uniformly good results with a minimum of trauma and postoperative care. It has been used in over one hundred consecutive cases by the author and the resident staff members with excellent results in the complete or partial restoration of the tympanic membrane.

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VIIeme INTERNATIONAL CONGRESS OF OTOLOGY, RHINOLOGY AND LARYNGOLOGY.

The Seventh International Congress of Otology, Rhinology and Laryngology will be held in Paris, July 23-29, 1961, at the New Faculte of Medicine, Rue des Saints-Peres. The program includes *1.* The Recent Acquisitions, Pathology and Therapeutics of Otosclerosis by Profs. Sourdille, Sercer and Krmpotic, Weber, Ardouin, Larroude, Ferreri and Shambaugh. *2.* Indications and Five-Year Results in Surgery and Radium in the Treatment of Cancers of the Larynx and Hypopharynx—Profs. Pietrantoni and Agazzi Alonzo, Casadesus, C. L. Jackson, Leicher, Leroux-Robert and Ormerod. *3.* A Report on Allergy and Infections of the Nose and Bronchus—Profs. Van Dishoeck, Clerici, Hlavacek, Voohorst, Mayer, Laskiewicz and Terracol.

Tours and entertainment for guests and their ladies have been arranged. President: Dr. Maurice Aubry; Secretary General: Henry Guillon. For further details write Dr. Guillon, 6 Avenue McMahon, Paris, France.

TWO RARE NASOPHARYNGEAL LESIONS: HODGKIN'S SARCOMA AND ONCOCYTOMA (Acidophil Adenoma).*

PAUL GUGGENHEIM, M.D.,

Council Bluffs, Ia.

The purpose of this communication is to report two rare nasopharyngeal lesions: Hodgkin's sarcoma and oncocytoma (or oxyphil adenoma). One patient exhibited both lesions. Although three cases of nasopharyngeal Hodgkin's[1-3] have been recorded (all in The Italian Literature, 1954-8), the present case was evidently the first to be encountered—during my first year of practice (1949). Oncocytoma, a rare tumor of salivary glands, has not been hitherto described in the nasopharynx. A patient exhibiting it was seen in 1959. Not until this experience brought the lesion to my attention, did a review of the slides from the 1949 Hodgkin's case reveal that this patient had a small nasopharyngeal focus of oncocytoma also.

Lautz,[4] describing nasal and laryngeal involvement in a case of abdominal Hodgkin's disease, remarks that " a number of articles presenting series of several hundred cases have little or no mention of upper respiratory tract involvement," and that "deafness, epistaxis and hoarseness are very infrequently presenting symptoms." He describes the case of a 26-year-old man with both nasal and laryngeal lesions. The nasopharynx was not described. A laryngeal biopsy showed only non-specific granuloma although at autopsy the typical Hodgkin's granuloma was demonstrated at this site as well as in the nose (not biopsied during life). The author warns that a biopsy reported as showing "non-specific granuloma" should make one think of Hodgkin's disease. The following case report will attest to the difficulty of establishing the true diagnosis in early lesions:

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*Dept. of Otolaryngology, Creighton University School of Medicine.

Editor's Note: This manuscript received in The Laryngoscope Office and accepted for publication Dec. 19, 1960.

CASE REPORT (Nasopharyngeal Hodgkin's Sarcoma).

A 66-year-old man was referred by his internist with complaints of recurring sore throat due to trench-mouth organisms, and of hearing impairment of two months' duration. The latter came on suddenly and was noted to be worse on the right.

Otoscopy revealed bilaterally retracted, glistening drums against which the ossicles were sharply skeletonized. No movement on pneumatic massage could be elicited on the right, and only a slight flap anteroinferiorly on the left, with no ossicular movement on either side. The Weber was lateralized to the right. Rinne's were negative bilaterally with 256, 512 and 1,024 forks. The Schwabach was prolonged on the right (21 sec.), compared with the left ear (16 sec.) and a control (the examiner, 16 sec.).*

The nose was normal. The mouth contained a complete upper and a partial lower denture. Tonsils were present and not remarkable. The right retrotonsillar lateral band appeared prominent when compared to its opposite number, and the right nasopharynx appeared "full," particularly in the region of the Rosenmueller fossa. On transillumination the frontal sinuses were clear but both antra considerably dimmed.

On the following day, having autoinflated himself with some success, the patient reported his ears improved although they looked unchanged. Reëxamination of the nasopharynx was noted as follows: "Full on right, can't see a *torus tubarius* as such. No ulceration." The right lingual tonsil was noted to be slightly enlarged. The larynx was normal. Examination of cranial nerves was negative except for a questionable slight fibrillary twitching of the right border of the tongue and a very slight meiosis of the right pupil. The neck showed two nodes in the deep anterior cervical chain on the right: one of these at the carotid bifurcation level, and two more behind the sternocleidomastoid. A collection of small shotty nodes was felt above the right clavicle.

A chest X-ray taken on November 19, 1949, by the internist (later reviewed by a radiologist) showed "mildly increased prominence in size of both hila but more of the type seen in upper respiratory infections."

On November 22, 1949, at the Los Angeles Eye and Ear Hospital a small horizontal incision was made over the mid portion of the right sternomastoid muscle. The muscle was split, and a large white, soft, friable node seen beneath the superior thyroid vein. The node broke during removal. The wound was electrocauterized and closed with drainage. A biopsy of the right nasopharynx was then done under local anesthesia with a scalpel and a Love palate retractor.

By December 3, the neck wound had healed. The remaining neck nodes had shown a definite regression. The supraclavicular nodes were no longer palpable. The mass in the right nasopharynx could hardly be distinguished. The patient insisted his hearing had returned to normal although the drums looked unchanged.

Meanwhile, the pathologist (who specialized in head and neck pathology) reported the specimens as follows:

"A—Tissue removed from the right nasopharynx consists of a long piece of mucosa 12 x 7 x 5 mm. and two smaller fragments. Microscopic examination shows a strip of mucous membrane with minor salivary glands affected by an irregularly focal chronic inflammatory reaction which contains principally lymphocytes and a few characteristic foam

*Audiometry was probably inaccurate with an obsolete Maico instrument badly in need of calibration.

cells. In one of the smaller pieces there is a small adenoma of minor salivary gland origin which is less than one microscopic field in size and which looks quite benign (see Fig. 1).

"B—Tissue removed from the region of the right deep cervical chain consists of a previously opened lymph node 19 x 15 x 12 mm. The cut surface has one scarred area. Miscroscopic examination shows a zone of fibrosis with diffuse chronic inflammatory reaction. The remainder of the node is involved by a hyperplastic process which has practically obliterated all of the normal architecture of the lymph node. This has

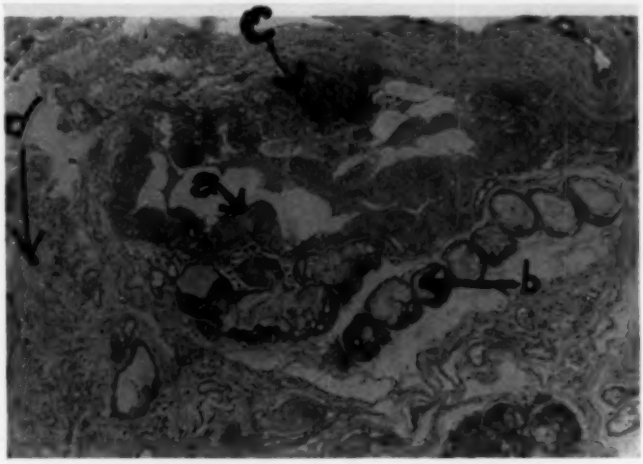

Fig. 1. Case 1. Original nasopharyngeal biopsy (November 22, 1949). No lymphoepithelioma seen in this specimen.
a. Small, single focus of oncocytoma with deep eosinophilic staining reaction.
b. Normal mixed mucous-serous glands of nasopharyngeal submucosa.
c. Small focus of non-malignant, inflammatory cells.
d. Loose collagenous connective tissue. (x 100)

a very variable pattern in which lymphocytes, plasma cells, foam cells and a few polynuclears are seen. There are some areas of this node which also contain a reticulum cell proliferation mixed with these other elements. A neoplasm cannot be definitely excluded (see Figs. 2 and 3).

"These sections have been shown to several other pathologists who agree that it is difficult to make an absolute diagnosis on this tissue.

"C—This tissue, removed along with B, consists of an irregular piece of lymphoid tissue 14 x 9 x 5 mm. submitted for bacteriological study. Bacteriological examination: The tissue was prepared by surface sterilization, maceration and emulsion. Smears contain no organisms. Acid-fast stain negative. Culture on blood agar plate sterile. Broth culture contains a putrefying nonfermenting, nonmotile gram negative bacillus which is probably a contaminant but which is being studied further.

Cultures for tubercle bacilli must be incubated for a minimum of three weeks and will be reported in six.

"DIAGNOSIS: A—Chronic pharyngitis.

B—Chronic hyperplastic lymphadenitis."

Comment: The fact that no suspicious Hodgkin's infiltrate was found in this nasopharyngeal specimen, the author ascribes his own inexperience in the surgery of the nasopharynx,

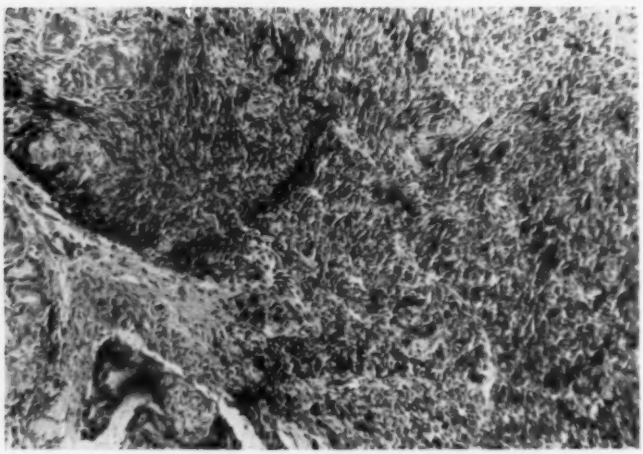

Fig. 2. Case 1. 2nd Biopsy, rt. nasopharynx, January 7, 1950, showing malignant infiltrate. (x 100)

for the presence of a nasopharyngeal tumor was the most striking feature of this case. The "small adenoma of minor salivary gland origin," which the pathologist did not further typify, was an oncocytoma and will be described more fully later.

Complement fixation tests for Neisseria and for lues (Kolmer) were negative, as were Kline and Kahn tests.

On December 12, only one enlarged node could be felt in the neck, in the middle of the right posterior cervical triangle; however, the patient was coming down with another sore throat. The pain of this was located in the right lateral nasopharyngeal and retrotonsillar oropharyngeal band, which was quite swollen and covered with a delicate gray exudate. The mass high in right nasopharynx remained small—now only a slight fullness. The laryngeal mirror, however, now revealed that the right retrotonsillar band extended downward into the right pyriform sinus. The

right arytenoid was of a deep reddish-purple color and so swollen as almost to obliterate the pyriform sinus.

A blood count on December 16, 1949, showed:

Hemoglobin—15.5 Gm.

RBC—5,340,000.

WBC—5,900.

Stab polynuclears—17%.

Mature polynuclears—52%.

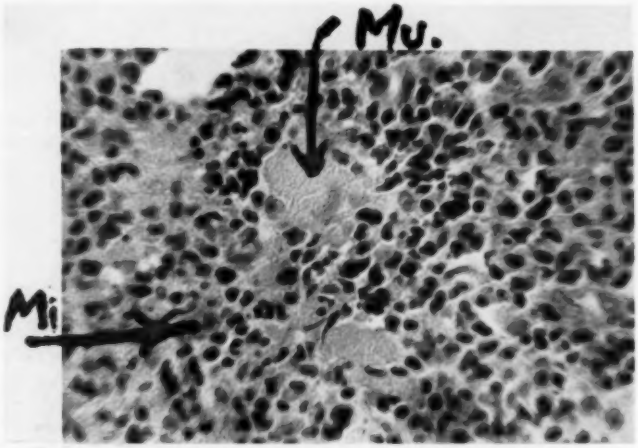

Fig. 3. Case 1. Biopsy of left posterior tonsillar pillar showing "lympho-epithelioma" invading muscle tissue (x 400). Mi = mitosis. Mu = muscle fibre.

Lymphocytes—19%.

Monocytes—7%.

Eosinophiles—5%.

A Frei test was negative. A culture from the lymph node removed on November 22, 1949, showed no acid fast organisms on Petragnani's medium after six weeks.

The recurring sore throat was retreated with penicillin and a culture taken for B. rhinoscleromatis. This was negative, but the positive results were as follows:

"Culture of throat: Cultures contain a few colonies of Streptococcus hemolyticus, some Neisseria catarrhalis, some coagulase negative Staphylococcus albus, and a number of colonies of Pseudomonas aeruginosa (B. pyocyaneus).

"Vincent's organisms were not seen in the direct smear of this material."

The pharyngeal membrane increased despite local (Abbott aerohaler)

and systemic penicillin and aureomycin, dilute hydrogen peroxide gargles and treatment by the internist with chromic acid.

On December 27, the patient called me to report that his neck was swollen on both sides, flush with the mandibles. This episode lasted only about three days and was due to an acute involvement of both submaxillary salivary glands and Wharton's ducts, exact nature unknown.

An X-ray of the petrous ridges taken on December 29 showed no erosions.

On December 31, examination with the Holmes electric nasopharyngoscope was recorded as follows: "White hypertrophy of the posterior tip of the right inferior turbinate. Behind this are glistening granulations which completely obscure the torus, extending onto the vault almost to the root of the septum."

A day or two later, an ulceration appeared on the anterior face of the left posterior tonsil pillar.

January 7, the original nasopharyngeal site (Rosenmueller fossa, right) and the left posterior pillar were biopsied. Under direct vision, a diffusely infiltrating, non-ulcerated lesion was seen in the nasopharynx. The soft palate was edematous. An ulcerated, fungating mass was present on the left posterior pillar. The right oropharyngeal gutter was almost filled by the very "hypertrophied lateral band." Both arytenoids were edematous. The pathologic report was "lymphoepithelioma" on both specimens (see Figs. 2 and 3).

An intensive program of radio-therapy was begun on January 17.

On January 16, the patient had a dark stool and felt weak. Blood count remained adequate. Bismuth was blamed and not a hemorrhage.

On January 19, chest X-ray was reported as showing a large oval-shaped mass measuring 4.0 x 6.0 Cm. in the lower two-thirds of the right hilum. The left hilum showed a uniform increased density. This picture was thought to show a definite change compared to its predecessor. Skull films again showed no bony erosions but cloudiness of all the paranasal sinuses and a "generalized increased density of the nasopharynx of soft-tissue type."

Despite vigorous therapy to the nasopharynx and neck through multiple ports, and to the mediastinum, the patient went progressively downhill, becoming febrile and deeply jaundiced, and expired on March 23, 1950.

An autopsy summary was as follows:

"*Revised Anatomical Diagnosis:*

Generalized icterus.

Emaciation.

Multiple keratoses and soft fibromas of the skin.

Old myocardial infarct involving the anterior wall, apex and anterior portion of the interventricular septum.

Occlusive atheromatous deposits of the branches of the anterior descending branch of the left coronary artery.

Extensive atheromatous change of the aorta and large arteries.

Hodgkin's infiltrates and ulceration of the mucosa of the nasopharynx.

Pleural effusion, right, 2,000 cc.

Fibrous pleuritis, left.

Atelectasis, right lower lobe of the lung.

Hodgkin's infiltration, focal, of the right lower lobe of the lungs.

Disseminated, periportal, Hodgkin's infiltrates of the liver.

Hodgkin's infiltrates in the wall of the gall bladder.

Hodgkin's infiltrates of the perilobular connective tissue of the pancreas.

Hodgkin's infiltrates and ulceration of the duodenum and ascending portion of the colon.

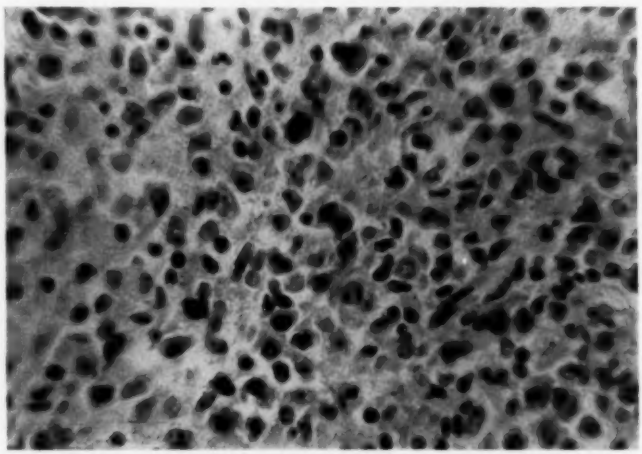

Fig. 4. Case 1. Periportal Hodgkin's infiltrate in liver (x 400). Several excellent examples of Reed-Sternberg cells to be seen.

Superficial ulceration of rectal mucosa.

Arteriosclerotic atrophy of the kidneys.

Cortical adenoma of the right kidney.

Nodular hyperplasia of the prostate gland.

Hodgkin's infiltrates of the spleen.

Perisplenitis.

Enlargement and Hodgkin's infiltrates of the cervical, axillary, substernal, paratracheal, tracheobronchial, bronchopulmonary and retroperitoneal lymph nodes.

Hodgkin's infiltrates of the adrenal cortices.

Chief Diagnosis:

Hodgkin's disease with extensive infiltration of the viscera and lymph nodes" (see Fig. 4).

Although the pathologist did not state the type of Hodgkin's represented, a recent review confirmed our opinion that this was a Hodgkin's sarcoma. There was virtually no fibrosis and no tissue eosinophilia, but a predominance of malignant reticulum cells of all sizes, including many typical Reed-Sternberg cells.

CASE REPORT: Oncocytoma (Acidophil Adenoma) of the right lateral Adenoid.

Ten years later a lanky, thin, arthritic, retired Iowa man of 68 was referred by his family physician because of pain in and about the right ear, unresponsive to chloromycetin therapy but unaccompanied by any fever. For a year or two the patient felt he had been losing hearing bilaterally, but particularly on the right. Otoscopy revealed the right drum to be diffusely bulging and putty-colored. Granulations protruded through a large attic perforation. The left drum was normal. The nose was negative except for darkness of both antra on transillumination. Tonsils were atrophic. Complete dentures were worn. The nasopharynx showed a moderate sized central adenoid mass covered with purulent exudate. A large right lateral band was noted which filled the fossa of Rosenmueller. No such band was seen on the left. The larynx showed a little pooling of saliva in the left pyriform sinus, presumably due to neurogenic weakness of the left constrictor. The neck was negative.

Past history revealed that at age 14 the patient had suffered a severe right-sided earache with spontaneous perforation of the drum followed by impaired hearing for two years. No further ear trouble was experienced for 50 years until, two years previously, *bilateral* hearing loss was first noted.

The patient had retired at 42 because of rheumatoid arthritis which began at age 27.

The present illness started abruptly with an intense pain in the forehead coming on at 10:00 A.M. on March 26, 1959. By afternoon "this pain had spread to the right ear." After 24 hours the patient experienced a severe vertiginous attack with nausea and vomiting which he and his doctor attributed to chloromycetin. Inquiry revealed that it persisted in milder form after discontinuation of the drug, so that his wife had to lead him about. When seen, he was suffering intensely, complaining that he felt with pain every pulse in his right ear and was aware of his extrasystoles.

X-rays of the paranasal sinuses were negative. Mastoid films showed normal structures on the left, well pneumatized. On the right, a mastoid of comparable extent showed marked clouding of the cells with fluid levels in many, and thickening of the intercellular bony septa.

On April 2, 1959, a right myringotomy was done and then an adenoidectomy was performed under local anesthesia. At some risk of lapse of dignity, proper to a case report, I am tempted to add a humorous note to illustrate the fact that an adenoidectomy under local anesthesia in an adult—even a very sick old gentleman—may be well borne with minimal discomfort. The patient had neglected to urinate before being brought to surgery, and throughout the operation somehow managed to carry on a vigorous conversation, urging the surgeon to hurry before his bladder burst, and telling off-color stories. After a meticulous removal of all

visible adenoid, he insisted on walking to the surgeons' lounge where, after much encouragement, he was able to empty his bladder and have a cup of coffee.

On April 10, he had a right attico-mastoidectomy. The findings indicated a long standing chronic process with much secondary sclerosis. Whether the disease experienced between ages 14 and 16 could have slumbered for 54 years, to be exacerbated by an acute adenoiditis, or whether a new disease arose phoenix-like from the extinct body of the old, one cannot say on the basis of pathologic or radiologic evidence alone.

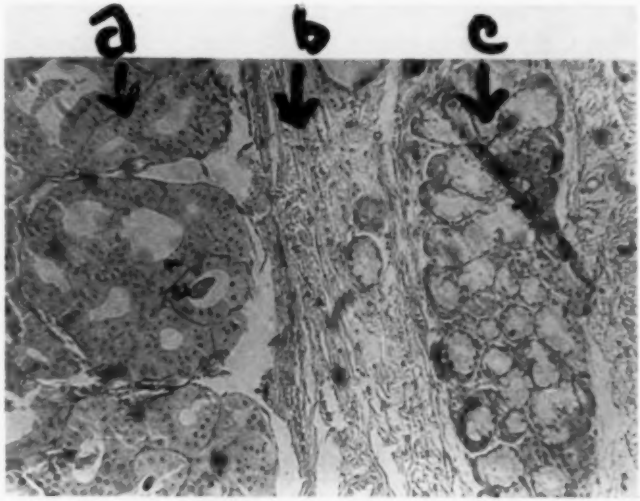

Fig. 5. Case 2. a. Oncocytoma separated from c) normal sero-mucinous glands of lateral-band submucosa by b) sheet of collagenous connective tissue. (x 100)

Recovery was complete and uneventful except for an allergic reaction to aureomycin in the form of a swollen tongue and loss of taste which slowly subsided.

Audiograms were taken between the two surgical procedures and subsequently and are shown in Table I.

The adenoid specimen showed nothing remarkable in the central mass, but the right lateral curtain showed a rather extensive oncocytoma (see Figs. 5 and 6). The role of this tumor in the pathogenesis of the recrudescent otitis-mastoiditis seems probable, in view of the extensive involvement of the torus and Rosenmueller fossa.

Oxyphil adenoma (or oncocytoma) is one of the rarer salivary gland tumors. According to Ackerman[5] it shares with

TABLE I.
Audiometric Data in Case of M.F.
(Oncocytoma, Rt. Lateral Adenoid)

Dates and Remarks	Right Ear										Left Ear									
	125	250	500	1000	1500	2000	3000	4000	6000	8000	125	250	500	1000	1500	2000	3000	4000	6000	8000
3/30/59 1st office visit AC-BC gap 15-20-35 db	40	40	45	55	60	80	85	—	—	—	20	10	5	5	20	40	65	70	65	45
4/ 6/59 After myringotomy and adenoidectomy (4/2/59)	45	35	25	30	35	40	80	70	—	—										
4/22/59 After attico-mastoid-ectomy (4/10/59)	25	20	15	40	55	60	85	—	—	—										
11/ 3/59	35	30	20	25	35	50	80	70	—	—	20	10	10	10	20	45	70	80	75	55

papillary cystadenoma lymphomatosum (Warthin's tumor) a common histogenesis from salivary duct epithelium. Neither tumor is prone to occur bilaterally. Oxyphil adenomata are said to grow slowly and not to attain large dimensions. They are composed of large cells with well-defined nuclei and granular cytoplasm. Mitotic figures are not seen. The recommended treatment is simple excision. Recurrence is said to be rare. Differential diagnosis between "hyperplasia" and

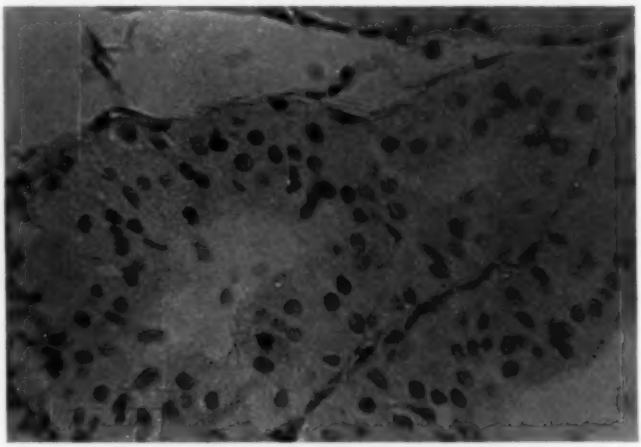

Fig. 6. Case 2. Oncocytoma-detail. (x 400)

"tumor" is noted to be difficult at times. Isolated cells of this type are said to increase with age and to occur most commonly in the parotid gland but also to have been seen in the submaxillary. Ackerman points out that the former gland shows considerable lymphoid tissue during embryogenesis, but not the latter.

No previous instance of this tumor in the nasopharynx has been found in the literature; however, when the slides of Case I were being reviewed, a small, isolated focus of oncocytoma in the original nasopharyngeal biopsy specimen was found; nor was this missed by the pathologist who originally

read the slide, although he did not specify the *type* of adenoma. Actually, such a lesion might be found wherever ectopic salivary gland tissue is found.

I would enter here a plea to consider most seriously the possibility of adenoid infection in adults, even the aged. I have found it by no means rare. Significant adenoid growths have been observed and removed in fully one-fourth of my adult tonsillectomies. Let anyone, who considers this percentage excessive ask himself how many nasopharynges of his adult tonsillectomy cases he has actually examined. Unless he has seen them all, he is in no position to discuss the question.

I would recommend, also, the direct transoral route for the biopsy of nasopharyngeal lesions. Direct inspection of the nasopharynx and adequate biopsy can be carried out speedily and efficiently under local anesthesia using a palate retractor and adenoid punch forceps, making unnecessary such tedious procedures as transnasal biopsy under mirror or nasopharyngoscopic guidance, and avoiding the palate-splitting approach to the nasopharynx advocated by Wilson[6] and others.

I have not encountered a case of juvenile nasopharyngeal fibroma, but only once, in my experience, was it found expedient to split the soft palate.

A Mexican boy of seven, one of triplets, presented a huge nasopharyngeal teratoma during my residency days at Children's Hospital, Los Angeles (1946-47). Alden Miller, assisted by Victor Goodhill and myself, simply split the soft palate longitudinally, removed the tumor with a cold snare, and sewed the palate up again using the method employed by our plastic colleagues. Had the tenuousness of the pedicle been known in advance, the division of the palate could have been avoided, for no bleeding whatever occurred, and the actual removal took approximately one minute.

SUMMARY.

1. A case of Hodgkin's sarcoma primary in the nasopharynx of an elderly man is presented. It is believed to be the first

such case observed chronologically, although the fourth to be reported. An incidental finding was a small focus of oncocytoma (acidophil adenoma) arising in the submucous glands of the involved area.

2. A second case of oncocytoma of the nasopharynx involving the lateral adenoid of an elderly man is described. This tumor of salivary glands has not been hitherto described in the nasopharynx.

3. Some technical considerations of nasopharyngeal biopsy are presented.

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TEMPLE UNIVERSITY SCHOOL OF MEDICINE AND HOSPITAL.

The next "Short Course in Audiology" will be presented by Temple University School of Medicine and Hospital June 19th through 23rd inclusive, 1961.

Further information may be obtained by writing to Philip E. Rosenberg, Ph.D., Director, Section of Audiology, Temple University Medical Center, 3401 North Broad St., Philadelphia, Penna.

PROGRAM OF THE
NINETY-FOURTH ANNUAL MEETING.
AMERICAN OTOLOGICAL SOCIETY, INC.

The Lake Placid Club — MAY 26 AND 27, 1961

Friday Morning, May 26, 1961.

Registration 8:00 A.M.

Presidential Remarks 8:30 A.M.

Introduction of Guests

Presentations.

1. The Differential Diagnosis of Vertigo Considered Bio-Anatomically.

   Benjamin Spector, M.D.

2. Studies on the Efferent Innervation of the Vestibular End Organs.

   Joseph Farkashidy, M.D.
   (By Invitation.)
   P. E. Ireland, M.D.

3. The Innervation Pattern of the Cochlear Hair Cells.

   Catherine A. Smith, Ph.D.
   (By Invitation.)
   Discussor: Hans Engstrom, M.D.

4. Pressures of the Labyrinthine Fluids No. 2.

   Francis L. Weille, M.D.
   John W. Irwin, M.D.
   (By Invitation.)
   Louise Clark, A.B.
   (By Invitation.)
   Phyllis Rahn, A.B.
   (By Invitation.)

5. Functional and Histologic Effects of Experimental Inner Ear Pressure Changes.

Brian F. McCabe, M.D.
(By Invitation.)
David A. Wolsk, Ph.D.
(By Invitation.)

Business Meeting.

Election of New Members.

Election of Nominating Committee.

Friday Afternoon, May 26, 1961.
2:00 P.M.

6. Circulation of the Fluids of the Inner Ear.

Merle Lawrence, Ph.D.

7. Some Vestibular Problems in Space Medicine.

Joseph A. Sullivan, M.B., F.R.C.S. (C)
Walter H. Johnson, Ph.D.
J. Brydon Smith, M.B., F.R.C.S. (C)
Discussor: Ralph Kraus, Col., MC, USA

8. The Effects of Kanamycin, Asphyxia, and Acoustic Trauma on the Permeability of the Cochlear Partitions.

Geo. Misrahy, M.D.
(By Invitation.)
Discussor: Joseph E. Hawkins, M.D.

9. The Effect of Sensorineural Lesions on Pitch Discrimination in Cats.

Donald Elliott, Ph.D.
(By Invitation.)
Harold F. Schuknecht, M.D.

10. Histopathologic Findings Following Stapes Surgery in the Human.

John R. Lindsay, M.D.

Friday Evening, May 26, 1961.

President's Reception 6:30 P.M.

Dinner 7:00 P.M.

For Members, Officially Invited Guests and Their Ladies—Dress Optional.

Saturday Morning, May 27, 1961.
9:00 A.M.

1. Stapedial, Capsular and Labyrinthine Anatomy in Relation to Otologic Surgery.
   Barry J. Anson, Ph.D.
2. A Clinical and Laboratory Evaluation of Polyethylene Tubing in Middle Ear Surgery.
   Francis A. Sooy, M.D.
3. Relation of Identification Audiometry and Otologic Conditions.
   Raymond E. Jordan, M.D.
   Eldon L. Eagles, M.D.
   (By Invitation.)
4. The Further Destruction of a Deafened Child's Hearing by the Use of Powerful Hearing Aids.
   Chas. E. Kinney, M.D.
   Discussor: S. Richard Silverman, Ph.D.

Recess 10 Minutes.

Business Meeting.

1. Minutes of Previous Annual Meeting.
2. Report of the Secretary-Treasurer.
3. Report of the Editor-Librarian.
4. Reports for the:
   a. Board of Trustees of the Central Bureau of Research.
      E. P. Fowler, M.D.
   b. American Standards Association.
      E. P. Fowler, M.D.

Saturday Noon, May 27, 1961.

c. American Board of Otolaryngology.
P. E. Meltzer, M.D.

d. The Award of Merit Committee.
Wm. J. McNally, M.D.

5. Recommendations of the Nominating Committee.

6. Reading of Communications.

7. Unfinished Business.

8. New Business.

Saturday Afternoon, May 27, 1961.
2:30 P.M.

5. Placebos, Antisludging Drugs and Disorders of the Ears.
Edmund Prince Fowler, M.D.

6. Age, Noise and Hearing Loss.
Aram Glorig, M.D.
Hallowell Davis, M.D.

7. Acoustic Measurements of Middle Ear Function.
Joseph Zwislocki, Ph.D.
(By Invitation.)

8. Animal Experiments on the Inner Ear (Moving Picture).
Albert Hohman, M.D.

CASSELBERRY AWARD OF THE AMERICAN LARYNGOLOGICAL ASSOCIATION.

A sufficient fund having accrued from the Casselberry Fund for encouraging advancement in the art and science of Laryngology and Rhinology, this sum is now available in part or as a whole, for a prize award. Theses must be in the hands of Dr. Lyman G. Richards, 12 Clovelly Road, Wellesley Hills 81, Mass., Secretary of the American Laryngological Association, prior to Dec. 1 of any given year.

The Award is a prize of money with accompanying certificate signed by the officers of the American Laryngological Association. The sum of money will be agreed upon by the Council of the Association after the manuscript has been evaluated by the Award Committee. It may be awarded in whole or in part among several contestants.

Eligible contestants may be: 1. Hospital interns, residents, or graduate students in Rhinology and Laryngology; 2. An individual with an M.D. degree who is actively practicing or teaching Rhinology and Laryngology in the Americas; 3. Any scientific worker in the field of Rhinology and Laryngology.

Manuscripts shall be presented to the Secretary of the Association under nom de plume which shall in *no way* indicate the author's identity. There shall also be a sealed envelope bearing the nom de plume and containing a card showing the name and address of the contestant which the Secretary shall keep in his possession.

Manuscripts must be limited to 5000 words and must be typewritten in double spacing on one side of the sheet. The thesis shall not have been published elsewhere before submission.

The successful thesis shall become the property of the American Laryngological Association, but this provision shall in no way interfere with publication of the thesis in the Journal of the author's choice. Unsuccessful contributions will be returned promptly to their authors.

The Award which will be made at the Annual Meeting of the American Laryngological Association shall be based on:

1. Originality of material.
2. Scientific and clinical value.
3. Suitability for this Award.
4. Method of presentation as to style, illustrations and reference.

The maximum amount of the Award shall not exceed $200.00.

THE WEST VIRGINIA ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY.

The West Virginia Academy of Ophthalmology and Otolaryngology will hold its annual meeting at the Greenbrier Hotel, White Sulphur Springs, W. Va., on April 6-8, 1961.

The guest speakers on Ophthalmology are: Dr. Irving H. Leopold, Philadelphia, Pa., and Dr. Harvey E. Thorpe, Pittsburgh, Pa.; for Otorhinolaryngology: Dr. John J. Shea, Memphis, Tenn., and Dr. F. Johnson Putney, Philadelphia, Pa.

In addition to the scientific program, arrangements have been made with Mr. Philip Salvatori of Obrig Laboratories to discuss and show techniques of contact lens fitting.

For additional information please contact the secretary, Dr. Worthy W. McKinney, 109 East Main St., Beckley, W. Va.

PROGRAM OF THE SIXTY-FOURTH ANNUAL MEETING.

AMERICAN LARYNGOLOGICAL, RHINOLOGICAL AND OTOLOGICAL SOCIETY, INC.

"THE TRIOLOGICAL SOCIETY."

Lake Placid Club, Essex County, New York.

MAY 23-25, 1961.

Time: **Tuesday, May 23, 1961.**

9:00 **Business Meeting.**

Election of the Nominating Committee.
Election of Candidates to Fellowship.
Induction of Newly Elected Fellows.

9:30 **Presidential Address.**

The Responsibility of the Otolaryngologist in the Prevention and Care of Automobile Injuries and Deaths.

Fletcher D. Woodward, M.D., Charlottesville, Va.

9:45 *E.N.T. in Re-ENT-ry.*

James Milton Robb, M.D., Detroit, Mich.
Guest of Honor.

Scientific Session.

10:15 1. *The Frequency and Severity of Injury to the Face and Jaws Associated with Automobile Accident Injuries.*

John O. Moore, Ph.D., Sayville, L. I., N. Y.
(By Invitation.)

10:35 Open Discussion.

10:45 Recess.

11:00 2. *The Immediate Repair of Injuries of the Face and Jaws.*
Edgar M. Holmes, M.D., Boston, Mass.

11:20 Open Discussion.

11:30 3. *Concepts in Facial Form and Function Rehabilitation.*
John J. Conley, M.D., New York, N. Y.

11:50 Open Discussion.

12:00 4. *Physiology and Pathology of the Cricothyroid Muscle* (Candidate's Thesis)
Godfrey E. Arnold, M.D., New York, N. Y.
No Discussion.

12:30 Adjournment.

Time: **Wednesday, May 24, 1961.**

9:00 **Business Meeting.**
Report of the Nominating Committee.
New and Miscellaneous Business.

9:20 *In Memoriam.*

Scientific Session.

9:30 5. *A Laboratory Study on Duration and Effect of IV Estrogen on Small Blood Vessels.*
Maurice Schiff, Captain, MC, USN.
H. Burn, M.S. (By Invitation.)
Both from U. S. Naval Hosp., Oakland, Calif.

9:50 Open Discussion.

10:00 6. *Problems of Weightlessness: Otolaryngological Aspects.*
Paul A. Campbell, Colonel, MC, USAF, Brooks Air Force Base, Texas.

10:20 Open Discussion.

10:30 Recess.

10:45 7. *Lymphatics of the Larynx and Neck.*
Joel J. Pressman, M.D., Los Angeles, Calif.

11:05 Open Discussion.

11:15 8. *Carcinoma of the Epiglottis—Importance of Early Diagnosis and Treatment.*

William A. Lell, M.D., Philadelphia, Pa.

11:35 Open Discussion.

11:45 9. *Functional Restitution of the Food Passage in Extensive Stenosing Caustic Burns of the Pharynx and Esophagus.*

Joseph H. Ogura, M.D.
Charles L. Roper, M.D. (By Invitation.)
Thomas H. Burford, M.D. (By Invitation.)
All of St. Louis, Mo.

12:05 Open Discussion.

12:15 Adjournment.

Time: **Thursday, May 25, 1961.**

9:00 **Business Meeting.**

Election of the Council.
Five-Minute Council Meeting.

Scientific Session.

9:30 10. *Variations in Normal Hearing: An Anatomical Explanation Based on Area-1 Ratio.*

William H. Saunders, M.D., Columbus, Ohio.
(By Invitation.)

9:50 Open Discussion.

10:00 11. *A Five-Year Report on Fenestration of the Oval Window with Vein Graft.*
(Illustrated with Color Movie.)

John J. Shea, M.D., Memphis, Tenn.
(By Invitation.)

10:20 Open Discussion.

10:30 Recess.

10:45 12. *Decompression of the Facial Nerve in Experimental Bell's Palsy in the Dog.*
Francis H. McGovern, M.D., Danville, Va.
J. S. Hansel, M.D., Danville, Va. (By Invitation.)

11:05 Open Discussion.

11:15 13. *Motion Picture Surgery of the Facial Nerve.*
George E. Shambaugh, Jr., M.D., Chicago, Ill.
Eugene L. Derlacki, M.D., Chicago, Ill.
Hollie E. McHugh, M.D., Montreal, Canada.
Jesse Waller, M.D., Chicago, Ill., and
Tawfik Girgis, M.D., Chicago, Ill. (Both by Invitation.)

11:45 Adjournment.

PENNSYLVANIA ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY.

The next annual meeting of the Pennsylvania Academy of Ophthalmology and Otolaryngology will be held May 18-19-20, 1961, at the Bedford Springs Hotel, Bedford, Pa.

The next annual meeting of the American Society of Ophthalmologic and Otolaryngologic Allergy will be held Saturday, October 7, 1961, at the Sherman Hotel, Chicago, Ill. The morning program will consist of a workshop on Diagnosis and Management of Allergic Disease. The afternoon program will consist of scientific presentations on Allergy, Immunity and Medical Otolaryngology.

The Twelfth Annual Eye and Ear Hospital of Pittsburgh Scientific Day will be Wednesday, April 19, 1961, at the Eye and Ear Hospital, Pittsburgh, Pa. The morning program will consist of a workshop on rhinoplasty and nasal and sinus diseases. The afternoon program will consist of scientific presentations in ophthalmology and otolaryngology.

DALLAS ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY.

PROGRAM 1960-1961.

May 2, 1961.

Parkland Memorial Hospital, Rooms No. 101-102.

5:30 P.M.—Clinical Otolaryngology and Ophthalmology Conference.

7:30 P.M.—Otolaryngology: Dr. William Wright, Houston, Tex. "Improved Methods of Handling Bony Deviations of the Nasal Septum." Discussion: Dr. Marvin G. Shepard, Dallas, Tex.

7:30 P.M.—Ophthalmology: "Non-Surgical Considerations in Horizontal Ocular Deviations." Dr. John Lippas, Parkland Memorial Hospital.

June 6, 1961.

Parkland Memorial Hospital, Rooms No. 101-102.

5:30 P.M.—Clinical Otolaryngology and Ophthalmology Conference.

7:30 P.M.—Otolaryngology: Papers presented by Residents of our Teaching Hospitals. Dr. Ben Stegal—"Pseudomonas Infections in Otolaryngology." Dr. Sam Huggins—"Experience in Rhinoplasty Procedures." Dr. Chris Helmus—"Aortic Hemograft Reconstruction in Radical Hypopharyngeal Surgery."

7:30 P.M.—Ophthalmology: "Ocular Problems Related to Space Medicine." Speaker to be announced.

SEVENTH INTERNATIONAL CONGRESS OF NEUROLOGY.

(Rome, 10-15 September 1961)

FIFTH INTERNATIONAL CONGRESS OF ELECTROENCEPHALOGRAPHY AND CLINICAL NEUROPHYSIOLOGY.

(Rome, 7-13 September 1961).

The Seventh International Congress of Neurology will be held in Rome, September 10-15, 1961, under the auspices of the World Neurologic Federation and of the National Institute for Nervous Diseases and Blindness of Bethesda.

Together with the general Assembly of the International League against Epilepsy and the V International Congress of Electroencephalography the first day will be dedicated to the opening Session.

Monday 11 and Tuesday 12 September will be given respectively to the discussion of the I and the II Topic, while the III Topic and the communications, will be represented respectively on Thursday 14 and Friday 15. Symposia will be held on Wednesday 13 September, a day free of other Congress engagements, to permit an interval between the two parts of the works.

Registration fees and other modalities are the same as those established for the Congress of Neurology. The official banquet and dance will be held in conjunction with this Congress.

Any information is obtainable from the President of the Congress, Prof. Mario Gozzano, or from the Secretary General, Dr. Raffaello Vizioli, Viale Universita 30, Roma.

UNIVERSITY OF TORONTO—POSTGRADUATE COURSE IN OTOLARYNGOLOGY.

On May 11, 12 and 13, 1961, there will be presented a graduate course in this specialty by the staff of the Department of Otolaryngology, assisted by two distinguished guests: Dr. Philip E. Meltzer, Interim Chief of Otolaryngology, Massachusetts Eye and Ear Infirmary, and Acting Head of the Department of Otolaryngology, Harvard Medical School; and Dr. W. G. Hemenway, Department of Otolaryngology, University of Chicago.

The first session will begin in the afternoon of May 11th, in the Royal York Hotel, Toronto, in conjunction with the Section of Otolaryngology of the Ontario Medical Association. The remainder of the sessions will be held in the clinical areas of the University of Toronto.

An attempt will be made to assess, discuss and demonstrate the newer procedures employed in the surgery of deafness. The present surgical treatment of head and neck problems will be presented with special consideration of the new conceptions of the responsibilities of our specialty in their management.

The fee for the course will be $40.00 and will include a complimentary dinner.

Please address all inquiries to the Director, Division of Postgraduate Medical Education, University of Toronto.

DIRECTORY OF OTOLARYNGOLOGIC SOCIETIES.

(Secretaries of the various societies are requested to keep this information up to date).

AMERICAN ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY.

President: Dr. Erling W. Hansen, 90 So. Ninth St., Minneapolis, Minn.
Executive Secretary: Dr. William L. Benedict, Mayo Clinic, Rochester, Minn.
Meeting: Palmer House, Chicago, Ill., October, 1961.

AMERICAN ASSOCIATION FOR CLEFT PALATE REHABILITATION.

President: Dr. J. J. Longacre, 1503 Carew Tower, Cincinnati, O.
President-Elect: Dr. D. C. Samuel Pruzansky, D.D.S., 840 So. Wood St., Chicago, Ill.
Secretary-Treasurer: Dr. Spriestersbach, Ph.D., Department of Otolaryngology, University Hospital, Iowa City, Ia.
Meeting:

AMERICAN BOARD OF OTOLARYNGOLOGY.

President: Dr. Gordon D. Hoople, 1100 E. Genesee Dr., Syracuse 10, N. Y.
Secretary: Dr. Dean M. Lierle, University Hospital, Iowa City, Ia.
Meeting: Palmer House, Chicago, Ill., October 2-5, 1961.

AMERICAN BRONCHO-ESOPHAGOLOGICAL ASSOCIATION.

President: Dr. F. Johnson Putney, 1712 Locust St., Philadelphia 3, Pa.
Vice-President: Dr. Julius W. McCall, 619 Rose Bldg., 2060 E. 9th St., Cleveland 15, O.
Secretary: Dr. Daniel C. Baker, Jr., 903 Park Ave., New York 21, N. Y.
Treasurer: Dr. Charles M. Norris, 3401 No. Broad St., Philadelphia 40, Pa.
Meeting: Lake Placid Club, Lake Placid, N. Y., May 23-24, 1961 (afternoons only).

AMERICAN LARYNGOLOGICAL ASSOCIATION.

President: Dr. Edwin N. Broyles, Baltimore, Md.
Secretary: Dr. Lyman G. Richards, Wellesley Hills, Mass.
Treasurer: Dr. Francis E. LeJeune, New Orleans, La.
Editor, Historian, and Librarian: Dr. Francis W. Davison, Danville, Pa.
Meeting: Lake Placid Club, Essex Co., N. Y., May 21-22, 1961.

AMERICAN LARYNGOLOGICAL, RHINOLOGICAL AND OTOLOGICAL SOCIETY, INC.

President: Dr. Fletcher D. Woodward, 400 Locust Ave., Charlottesville, Va.
President-Elect: Dr. John R. Lindsay, Chicago, Ill.
Secretary: Dr. C. Stewart Nash, 700 Medical Arts Bldg., Rochester 7, N. Y.
Treasurer: Dr. K. M. Day, 121 University Pl., Pittsburgh, Pa.
Annual Meeting: Lake Placid Club, Essex Co., N. Y., May 23-24-25, 1961.

AMERICAN MEDICAL ASSOCIATION, SECTION ON LARYNGOLOGY, OTOLOGY AND RHINOLOGY.

Chairman: Dr. Lawrence R. Boies, Minneapolis, Minn.
Vice-Chairman: Dr. Howard P. House, Los Angeles, Calif.
Secretary: Dr. Walter E. Heck, San Francisco, Calif.
Delegate: Dr. Gordon F. Harkness, Davenport, Ia.
Alternate Delegate: Dr. Paul H. Holinger, Chicago, Ill.
Representative to Scientific Exhibit: Dr. Walter H. Maloney, Cleveland, O.
Meeting: New York City, June 26-30, 1961.

AMERICAN OTOLOGICAL SOCIETY, INC.

President: Dr. Henry L. Williams, Rochester, Minn.
Vice-President: Dr. Lawrence R. Boies.
Secretary-Treasurer: Dr. James A. Moore, New York City, N. Y.
Annual Meeting: Lake Placid Club, Essex Co., N. Y., May 26-27, 1961.

AMERICAN OTORHINOLOGIC SOCIETY FOR THE ADVANCEMENT OF PLASTIC AND RECONSTRUCTIVE SURGERY.

President: Dr. Joseph Gilbert, 111 E. 61st St., New York, N. Y.
Vice-President: Dr. Kenneth Hinderer, 402 Medical Arts Bldg., Pittsburgh, Pa.
Secretary: Dr. Louis Joel Feit, 66 Park Ave., New York 16, N. Y.
Treasurer: Dr. Arnold L. Caron, 36 Pleasant St., Worcester, Mass.

AMERICAN RHINOLOGIC SOCIETY.

President: Dr. Ivan W. Philpott, 1801 High St., Denver, Colo.
Secretary: Dr. Robert M. Hansen, 2210 Lloyd Center, Portland 12, Ore.
Annual Clinical Session: Chicago, Ill., October, 1961 (definite time and place to be announced later).
Annual Meeting: Chicago, Ill., October, 1961 (definite time and place to be announced later).

AMERICAN SOCIETY FOR HEAD AND NECK SURGERY.

President: Dr. John J. Conley, New York, N. Y.
Vice-President: Dr. Joseph H. Ogura, St. Louis, Mo.
Treasurer: Dr. F. Johnson Putney, Philadelphia, Pa.
Secretary: Dr. George A. Sisson, Syracuse, N. Y.
Annual Meeting: Palmer House, Chicago, Ill.

AMERICAN SOCIETY OF FACIAL PLASTIC SURGERY.

President: Dr. Sam H. Sanders, 1089 Madison Ave., Memphis, Tenn.
Vice-President: Dr. John T. Dickinson, Mercy Hospital, Pittsburgh 19, Pa.
Secretary: Dr. Samuel M. Bloom, 123 East 83rd St., New York 28, N. Y.
Treasurer: Dr. Joseph C. Miceli, 879 Glenmore Ave., Brooklyn, N. Y.
Winter Meeting: February 9-10, 1961, Peabody Hotel, Memphis, Tenn.
Summer Meeting: June 28, 1961, Hotel Elysee, New York, N. Y.

AMERICAN SOCIETY OF OPHTHALMOLOGIC AND OTOLARYNGOLOGIC ALLERGY.

President: Dr. Leland H. Prewitt, Ottumwa, Ia.
Secretary-Treasurer: Dr. Daniel S. DeStio, 121 S. Highland Ave., Pittsburgh 6, Pa.
Annual Meeting: Palmer House, Chicago, Ill., October 7, 1961.

ASSOCIACAO MEDICA DO INSTITUTO PENIDO BURNIER—CAMPINAS.

President: Dr. Alberto Gallo.
First Secretary: Dr. Alfredo Martinelli.
Second Secretary: Dr. Guedes de Melo Neto.
Librarian-Treasurer: Dr. L. de Souza Queiroz.
Editors for the Archives of the Society: Dr. Antonio de Almeida, Dr. Gabriel Pôrto, and Dr. Roberto Franco do Amaral.

ASOCIACION DE OTORRINOLARINGOLOGIA Y BRONCOESOFAGOLOGIA DE GUATEMALA.

Presidente: Dr. Julio Quevedo, 15 Calle Oriente No. 5.
First Vice-Presidente: Dr. Héctor Cruz, 3a Avenida Sur No. 72.
Second Vice-Presidente: Dr. José Luis Escamilla, 5a Calle Poniente No. 48.
Secretario-Tesorero: Dr. Horace Polanco, 13 Calle Poniente No. 9-D.

ASOCIACION DE OTO-RINO-LARINGOLOGIA DE BARCELONA, SPAIN.

Presidente: Dr. J. Abello.
Vice-Presidente: Dr. Luis Suñe Medan.
Secretario: Dr. Jorge Perellò, 319 Provenza, Barcelona.
Vice-Secretario: Dr. A. Pinart.
Vocal: Dr. J. M. Ferrando.

BALTIMORE NOSE AND THROAT SOCIETY.

Chairman: Dr. Walter E. Loch, 1039 No. Calvert St., Baltimore, Md.
Secretary-Treasurer: Dr. Theodore A. Schwartz.

BUENOS AIRES CLUB OTOLARINGOLOGICO.

Presidente: Dr. K. Segre.
Vice-Presidente: Dr. A. P. Belou.
Secretario: Dr. S. A. Aranz.
Pro-Secretario: Dr. J. M. Tato.
Tesorero: Dr. F. Games.
Pro-Tesorero: Dr. J. A. Bello.

CANADIAN OTOLARYNGOLOGICAL SOCIETY SOCIETE CANADIENNE D'OTOLARYNGOLOGIE.

President: Dr. Fernand Montreuil, 1123 St. Joseph Blvd. East, Montreal, Quebec.
Secretary: Dr. Donald M. MacRae, 324 Spring Garden Road, Halifax, Nova Scotia.
Meeting: Queen Elizabeth Hotel, June 15-17, 1961.

CENTRAL ILLINOIS SOCIETY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY.

President: Dr. G. E. Hartenbower, 203 N. Main St., Bloomington, Ill.
President-Elect: Dr. Edgar T. Blair, Springfield, Ill.
Vice-President: Dr. G. LeRoy Porter, Urbana, Ill.
Delegate at Large: Dr. S. G. Baldwin, Danville, Ill.
Secretary-Treasurer: Dr. C. L. Pannabecker, Peoria, Ill.

CHICAGO LARYNGOLOGICAL AND OTOLOGICAL SOCIETY.

President: Dr. George Woodruff, Woodruff Clinic, Joliet, Ill.
Vice-President: Dr. Linden Wallner, 122 So. Michigan, Chicago, Ill.
Secretary-Treasurer: Dr. Robert Lewy, 25 East Washington St., Chicago 2, Ill.
Meeting: First Monday of each month, October through May.

CHILEAN SOCIETY OF OTOLARYNGOLOGY.

President: Dr. Enrique Grünwald S.
Vice-President: Dr. Agustin Estartus.
Secretary: Dr. Marcos Chaimovich S.
Treasurer: Dr. Benjamin Kapkan K.
Director: Dr. Alberto Basterrica A.

COLORADO OTOLARYNGOLOGY SOCIETY.

President: Dr. James T. Blair, Denver, Colo.
Vice-President: Dr. James Rigg, Grand Junction, Colo.
Secretary: Dr. Will P. Pirkey, Denver, Colo.

COLUMBUS, OHIO, OPHTHALMOLOGICAL AND OTOLARYNGOLOGICAL SOCIETY.

President: Dr. John E. Arthur.
Secretary: Dr. M. L. Battles.
Meetings: First Monday of October through May, University Club, Columbus, O.

DALLAS ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY.

President: Dr. Edward A. Newell.
Vice-President: Dr. Thomas M. McCrory.
Secretary-Treasurer: Dr. James L. Baldwin, 1627 Medical Arts Bldg., Dallas, Tex.

FEDERACION ARGENTINA, DE SOCIEDADES DE OTORRINOLARINGOLOGIA.

Secretary of the Interior: Prof. Dr. Atilio Viale del Carril.
Secretary of the Exterior: Dr. Aldo G. Remorino.
Secretary Treasury: Prof. Dr. Antonio Carrascosa.
Pro-Secretary of the Interior: Prof. Dr. Carlos P. Mercandino.
Pro-Secretary of the Exterior: Prof. Dr. James A. del Sel.
Pro-Secretary of the Treasury: Dr. Jorge Zubizarreta.

FIRST CENTRAL AMERICAN CONGRESS OF OTORHINOLARYNGOLOGY.

President: Dr. Victor M. Noubleau, San Salvador.
Secretary-Treasurer: Dr. Hector R. Silva, Calle Arce No. 84, San Salvador, El Salvador, Central America.

FLORIDA SOCIETY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY.

President: Dr. G. Dekle Taylor, Jacksonville, Fla.
President-Elect: Dr. Kenneth S. Whitmer, Miami, Fla.
First Vice-President: Dr. William H. Anderson, Jr., Ocala, Fla.
Second Vice-President: Dr. Marion W. Hester, Lakeland, Fla.
Secretary-Treasurer: Dr. Joseph W. Taylor, Jr., 1 Davis Blvd., Tampa 6, Fla.

FOURTH LATIN-AMERICAN CONGRESS OF OTORINOLARINGOLOGIA.

President: Dr. Dario.
Secretary:
Meeting:

FORT WORTH EYE, EAR, NOSE AND THROAT SOCIETY.

President: Dr. Van D. Rathgeber.
Vice-President: Dr. William Skokan.
Secretary-Treasurer: Dr. Paul Rockwell.

GEORGIA SOCIETY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY.

President: Dr. P. W. Rhyne, Albany, Ga.
Vice-President: Dr. C. L. Pennington, Macon, Ga.
Secretary: Dr. James T. King, 516 Baptist Professional Bldg., Atlanta 12, Ga.
Meeting: Oglethorpe Hotel, Wilmington Island, Savannah, Ga., March 2-4, 1961.

GREATER MIAMI EYE, EAR, NOSE AND THROAT SOCIETY.

President: Dr. Mariano C. Caballero.
Vice-President: Dr. Joseph Freeman.
Secretary-Treasurer: Dr. H. Carlton Howard.
Meeting: Quarterly in March, May, October and December on the second Thursday of the month, 6:30 P.M., at the McAllister Hotel, Miami, Fla.

INTERNATIONAL BRONCHOESOPHAGOLOGICAL SOCIETY.

President: Dr. Jo Ono, Tokyo, Japan.
Secretary: Dr. Chevalier L. Jackson, 3401 N. Broad St., Philadelphia 40, Pa., U. S. A.
Meeting:

KANSAS CITY SOCIETY OF OTOLARYNGOLOGY AND OPHTHALMOLOGY.

President: Dr. Clarence H. Steele.
President-Elect: Dr. Dick H. Underwood.
Secretary: Dr. James T. Robison, 4620 J. C. Nichols Parkway, Kansas City, Mo.
Meeting: Third Thursday of November, January, February and April.

LOS ANGELES SOCIETY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY.

President: Dr. Max E. Pohlman.
Secretary-Treasurer: Dr. Wendell C. Irvine.
Chairman of Ophthalmology Section: Dr. Carroll A. McCoy.
Secretary of Ophthalmology Section: Dr. Philip D. Shanedling.
Chairman of Otolaryngology Section: Dr. Robert W. Godwin.
Secretary of Otolaryngology Section: Dr. Francis O'N. Morris.
Place: Los Angeles County Medical Association Bldg., 1925 Wilshire Blvd., Los Angeles, Calif.
Time: 6:30 P.M. last Monday of each month from September to June, inclusive—Otolaryngology Section. 6:30, first Thursday of each month from September to June, inclusive—Ophthalmology Section.

LOUISIANA-MISSISSIPPI OPHTHALMOLOGICAL AND OTOLARYNGOLOGICAL SOCIETY.

President: Dr. Arthur V. Hays.
Secretary: Dr. Edley H. Jones, 1301 Washington St., Vicksburg, Miss.
Meeting: Edgewater Gulf Hotel, Edgewater Park, Miss., May 12-13, 1961.

MEMPHIS SOCIETY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY.

Chairman: Members serve as chairman in alphabetical order monthly.
Secretary-Treasurer: Dr. Roland H. Myers, 1720 Exchange Bldg., Memphis, Tenn.
Assistant Secretary-Treasurer: Dr. William F. Murrah, Jr., Exchange Bldg., Memphis, Tenn.
Meeting: Second Tuesday in each month at 8:00 P.M. at Memphis Eye, Nose and Throat Hospital.

MEXICAN ASSOCIATION OF PLASTIC SURGEONS.

President: Dr. Cesar LaBoide, Mexico, D. F.
Vice-President: Dr. M. Gonzales Ulloa, Mexico, D. F.
Secretary: Dr. Juan De Dios Peza, Mexico, D. F.

MEXICAN SOCIETY OF OTOLARYNGOLOGY.

President: Dr. Rafael Giorgana.
Secretary: Dr. Carlos Valenzuela, Petrarca 332-1, Mexico 5, D. F.

THE MINNESOTA ACADEMY OF OPHTHALMOLOGY AND OTO-LARYNGOLOGY.

President: Dr. Paul G. Bunker, Aberdeen, S. Dak.
First Vice-President: Dr. John A. McNeill, St. Paul, Minn.
Second Vice-President: Dr. Benjamin Bofenkamp, Minneapolis, Minn.
Secretary-Treasurer: Dr. Richard O. Leavenworth, Jr., St. Louis Park, Minn.

MISSISSIPPI VALLEY MEDICAL SOCIETY.

President: Dr. Arthur S. Bristow, Princeton, Mo.
Secretary-Treasurer: Dr. Harold Swanberg, Quincy, Ill.
Assistant Secretary-Treasurer: Dr. Jacob E. Reisch, Springfield, Ill.

NETHERLANDS SOCIETY OF OTO-RHINO-LARYNGOLOGY.
(Nederlandsche Keel-Neus-Oorheelkundige Vereeniging.)

President: Dr. H. Navis, Sonsbeekweg 6, Arnhem.
Secretary: Dr. W. H. Struben, J. J. Viottastraat 1, Amsterdam.
Treasurer: Mrs. F. Velleman-Pinto, Jac. Ohrechtstr. 66, Amsterdam.

NORTH CAROLINA EYE, EAR, NOSE AND THROAT SOCIETY.

President: Dr. George T. Noel, Kannapolis, N. C.
Vice-President: Dr. J. C. Bradley, Weaverville, N. C.
Secretary: Dr. John S. Gordon, 1350 South Kings Drive, Charlotte, N. C.

NORTH OF ENGLAND OTOLARYNGOLOGICAL SOCIETY.

President: Mr. G. L. Thompson, 16 Ramshill Road, Scarborough, Yorkshire.
Vice-President: Mr. J. H. Otty, Frizley Old Hall, Frizinghall Road, Bradford, Yorkshire.
Secretary and Treasurer: Mr. R. Thomas, 27 High Petergate, York, Yorkshire.

OREGON ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY.

President: Dr. George C. Saunders, 644 Medical Arts Bldg., Portland 5, Ore.
Secretary-Treasurer: Dr. Donald C. Mettler, 1216 S. W. Yamhill St., Portland 5, Ore.
Meeting: Fourth Tuesday of each month from September through May, Aero Club, Portland, Ore.

OTOSCLEROSIS STUDY GROUP.

President: Dr. Arthur L. Juers, 1018 Brown Bldg., Louisville, Ky.
Vice-President: Dr. Victor Goodhill, 4759 Hollywood Blvd., Los Angeles 27, Calif.
Secretary-Treasurer: Dr. R. E. Jordan, 3515 Fifth Ave., Pittsburgh 13, Pa.
Meeting: Palmer House, Chicago, Ill., October, 1961.

PACIFIC COAST OTO-OPHTHALMOLOGICAL SOCIETY.

President: Dr. John F. Tolan, 1118-9th Ave., Seattle 5, Wash.
Secretary-Treasurer: Dr. Homer E. Smith, 686 Twelfth Ave., Salt Lake City, Utah.

PAN AMERICAN ASSOCIATION OF OTO-RHINO-LARYNGOLOGY AND BRONCHO-ESOPHAGOLOGY.

President: Dr. Paul Holinger, 700 No. Michigan Blvd., Chicago, Ill.
Executive Secretary: Dr. Chevalier L. Jackson, 3401 No. Broad St., Philadelphia 40, Pa., U. S. A.
Meeting: Seventh Pan American Congress of Oto-Rhino-Laryngology and Broncho-Esophagology.

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